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STUDIES IN TUBERCULOSIS. I: THE INCIDENCE OF TUBERCULOSIS INFECTION IN AND ABOUT SYDNEY, NEW SOUTH WALES, AS SHOWN BY THE INTRACUTANEOUS TUBERCULIN TEST.[1]

By DOUGLAS ANDERSON, M.D., M.R.C.P., F.R.A.C.P.,

Honorary Assistant Physician, Royal North Shore Hospital of Sydney.

The Object, Method and Scope of the Investigation.

DURING the year 1938 a survey was made to determine the incidence of tuberculosis infection (as distinct from manifest tuberculous disease) in and about Sydney, a city of about 1,200,000 inhabitants. The intracutaneous tuberculin test of Mantoux was employed for this purpose, the incidence of tuberculosis infection and tuberculin sensitization being regarded as equivalent epidemiologically. The tuberculin employed was the purified protein derivative (tuberculin P.P.D.). A new technique for carrying out the test was devised and adopted and has already been described.(1) The plan of the survey was to carry out Mantoux tests of a large number of people who would represent a fair sample of the population. All the persons tested were apparently healthy, but those few who happened to have lived in tuberculosis households were not excluded. Relatively many more tests were made of young people than of old people; but persons in all walks of life were tested and the numbers amounted to 0·5% of the entire population of Sydney.

General Analysis of Results.

The results of the tests of 5,822 subjects are shown in Table I, which sets out the number of

[1] Work carried out with the aid of a grant from the National Health and Medical Research Council of the Commonwealth of Australia. From the Royal North Shore Hospital of Sydney Institute of Medical Research. Submitted as a thesis for the degree of Doctor of Medicine in the University of Sydney, January 7, 1939.

TABLE I.

Reactions to the Intracutaneous Tuberculin Test of 5,822 apparently Healthy Persons living in and about Sydney.

Ages.	Males.			Females.		
	Number Tested.	Number of Reactors.	Reactors *per centum.*	Number Tested.	Number of Reactors.	Reactors *per centum.*
0 to 4	55	3	5·5	73	5	6·8
5 to 9	254	29	11·4	273	40	14·7
10 to 14	494	112	22·7	547	113	20·7
15 to 19	554	200	36·1	697	204	29·3
20 to 24	556	322	57·9	852	412	48·4
25 to 29	294	203	69·0	358	219	61·2
30 to 34	165	119	72·1	148	102	68·9
35 and over	336	288	85·7	166	117	70·5
Totals	2,708			3,114		

persons of each sex tested in each quinquennial age group and the proportion of reactors. Figure I is a graphic representation of this table.

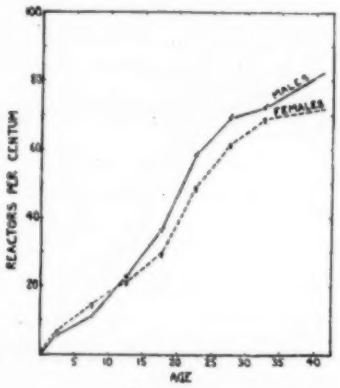

FIGURE I.

Reactions to the intracutaneous tuberculin test of 5,822 apparently healthy persons grouped according to age and sex. (From Table I.)

That the random sample of the population was a fairly representative one and that the technique of the test and its interpretation were uniform is indicated by the approximation of the proportion of reactors, age for age, in five series, each of about 1,000 consecutive subjects (Table II and Figure II).

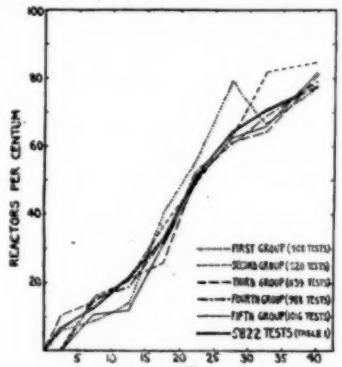

FIGURE II.

Reactions to the intracutaneous tuberculin test of five different groups, each of about 1,000 consecutive subjects. The approximation of the proportion of reactors, age for age, in each group shows that the random sample of the population was fairly representative and the technique and interpretation of the test were uniform. (From Table II.)

TABLE II.

Comparison of the Reactions to the Intracutaneous Tuberculin Test of Five Series each of about 1,000 Consecutive Subjects.

Ages.	First Group.			Second Group.			Third Group.			Fourth Group.			Fifth Group.		
	Number Tested.	Number of Reactors.	Reactors *per centum.*	Number Tested.	Number of Reactors.	Reactors *per centum.*	Number Tested.	Number of Reactors.	Reactors *per centum.*	Number Tested.	Number of Reactors.	Reactors *per centum.*	Number Tested.	Number of Reactors.	Reactors *per centum.*
0 to 4 ..	52	3	5·8	24	0	0·0	20	2	10·0	4	0	0·0	10	0	0·0
5 to 9 ..	107	10	9·3	51	8	15·7	215	32	14·8	70	11	15·7	19	2	10·5
10 to 14 ..	57	8	14·0	39	8	20·6	544	113	20·8	322	59	18·3	34	4	11·8
15 to 19 ..	122	49	40·2	245	91	37·1	176	45	25·6	261	85	32·6	299	94	31·4
20 to 24 ..	343	197	57·4	227	114	50·0	180	94	52·2	173	93	53·8	318	162	50·9
25 to 29 ..	147	117	79·6	96	61	63·5	79	48	60·8	83	50	60·3	125	79	63·2
30 to 34 ..	54	36	66·7	55	45	81·8	14	9	64·3	38	26	68·4	67	44	65·6
35 and over	26	21	80·8	83	71	85·5	21	18	85·7	37	30	81·8	144	125	86·8
Totals ..	908			820			1,139			988			1,016		

TABLE III.

Comparison of Several Recent Surveys of the Incidence of Tuberculosis Infection in the General Populations by means of the Intracutaneous Tuberculin Test.

Ages.	London, 1929.[2]		U.S.A., 1935.[3]		Sydney, 1938.		Ages.
	Number Tested.	Reactors *per centum.*	Number Tested.	Reactors *per centum.*	Number Tested.	Reactors *per centum.*	
0 to 2	121	7	842	13	128	6	0 to 4
3 to 5	124	18					
6 to 10	245	33	7,682	19	527	13	5 to 9
11 to 20	127	65	15,124	31	1,041	22	10 to 14
			17,097	34	1,251	32	15 to 19
21 and over	134	93	7,117	54	1,408	52	20 to 24

Comparison with Similar Surveys in other Countries.

In order to provide a standard for comparison with these analyses, Table III (illustrated by Figure III) has been included. This table sets out similar analyses made in recent years by Hart,[2] who carried out 751 Mantoux tests of non-tuberculous young people in London in 1929, and by Whitney and McCaffrey,[3] who tabulated the results of 56,688 Mantoux tests carried out in various parts of the United States of America under the auspices of the American National Tuberculosis Association. It is seen that at all ages the population of Sydney is much less heavily infected than that of London. The population of Sydney is also less heavily infected in childhood than the American populations, although after the age of twenty the proportion of those infected is about equal in the two populations. In Figure IV a similar comparison is made of the present survey with a survey carried out in Oslo recently, and mentioned by Heimbeck.[4] Heimbeck does not give his figures, and the curves in Figure IV are copied as accurately as possible from his graphs. The correspondence of infection by tuberculosis of the population of Sydney with that of the "middle classes" of Oslo is very close. It is of some importance to point out this parallel, because in Oslo at present much work is being carried on in the epidemiology of tuberculosis, and the conclusions which we may expect to be published may have an application to our own population. It must be borne in mind, however, that manifest tuberculosis is at least three times as prevalent in Scandinavia as in Australia,[5] and most of the groups studied by the cutaneous tuberculin test appear to be much more heavily and early infected.

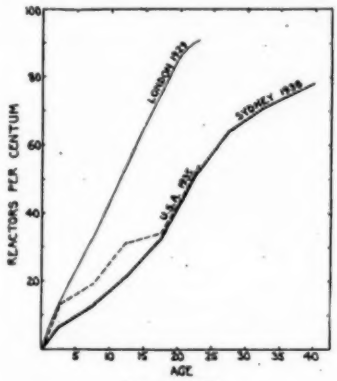

FIGURE III.

Comparison of the present survey with two other recent similar surveys. (From Table III.)

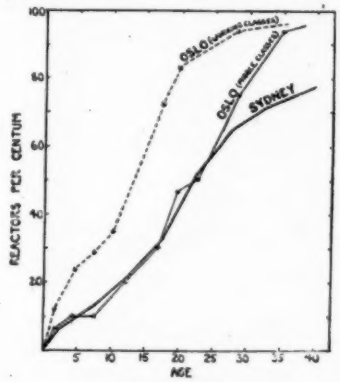

FIGURE IV.

Comparison of the present survey with that made in Oslo recently. (The Oslo curves are copied from the graphs of Heimbeck.[4])

Comparison of the Sexes.

Males and females are found to be infected almost equally in Sydney in the years of childhood, the proportion of girls being slightly the higher. Between the ages of ten and fourteen, boys are somewhat more prone to become infected than girls. This trend is even more evident between the ages of fifteen and nineteen, the proportion of reacting males being nearly doubled, while the proportion of reacting females is increased only by half. In the quinquennium between the ages of twenty and twenty-four the curve of infection rises steeply but about equally for both sexes. Between the ages of twenty-five and twenty-nine,

when many women are returning to domesticity, there is a slight falling off of the increase of infection among females, but the proportion of males becoming infected rises as steeply as before. After the age of thirty the curves of infection become parallel once more and much less steep. After the age of thirty-five, 85% of male subjects were found to react to the tuberculin test and only 74% of female subjects. It may be deduced that when tuberculosis infection has not been contracted in childhood from domestic sources it is most apt to occur when the young person is a good deal away from the sheltered environment of the home and is making his or her own way in the world.

That tuberculization varies inversely with the degree of domesticity is again suggested by the tuberculin reactions (set out in Table IV) of 519 young women, aged fifteen to nineteen years, of whom 248 were workers or undergraduates, while 271 had never had to earn their living. The subjects upon whose tuberculin reactions this finding is based, come, of course, from non-tuberculosis households. The special susceptibility of some members of tuberculous families to develop tuberculous infection and disease is not denied; nor is the desirability of genealogical investigation in every case of tuberculous disease.

TABLE IV.

Reactions to the Intracutaneous Tuberculin Test of 519 Young Women aged fifteen to nineteen years, some Workers and some who had always Lived at Home.

Workers.			Always Lived at Home.		
Number Tested.	Number of Reactors.	Reactors *per centum.*	Number Tested.	Number of Reactors.	Reactors *per centum.*
248	83	33·5	271	69	25·4

Home Contacts and Non-Contacts.

It is now common knowledge that a much larger proportion of persons coming from tuberculous households ("home contacts") reacts to the tuberculin test than of those coming from non-tuberculous households ("non-contacts"). Figure V gives a graphic comparison of the tuberculin reactions of the present series of 5,822 persons with those of 1,133 patients under the care of Dr. Cotter Harvey at the Royal North Shore Hospital of Sydney, who came from tuberculous households but were not themselves clinically tuberculous. The reactions of these persons are also analysed in Table V. The proportions given in this table bear a very close resemblance to those given by Whitney and McCaffrey[3] in a survey of 8,276 subjects from tuberculous households in the United States of America in 1935, except that the proportions infected in Sydney are a little higher. The great access of infection in infancy and childhood in those who come from tuberculous households is apparent, 40% of those under the age of five years being infected, whereas only 6% of those under the age of five years who do not come from tuberculous households are infected. After the age of five years and up to the age of fifteen years the contacts remaining uninfected become infected at the rate of about 18% per quinquennium, and the non-contacts at the rate of about 10% per quinquennium. Thereafter the respective liability to infection of the two groups is about equal.

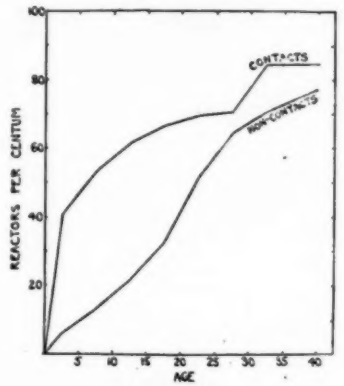

FIGURE V.

Reactions to the intracutaneous tuberculin test of 1,133 contacts and 5,822 non-contacts, grouped according to age. (From Tables V and I.)

TABLE V.

Reactions to the Intracutaneous Tuberculin Test of 1,133 Persons from Tuberculous Households.

Ages.	Number Tested.	Number of Reactors.	Reactors *per centum.*
0 to 4	122	49	40·2
5 to 9	214	115	53·7
10 to 14	227	141	62·1
15 to 19	112	75	67·0
20 to 24	92	64	69·6
25 to 29	77	54	70·1
30 to 34	84	71	84·5
35	205	173	84·4
Total	1,133		

Age of First Infection.

It has been shown that in Sydney a relatively large proportion of young people first encounter the tubercle bacillus at the ages when they are leaving home and going out into the world; in the other populations represented in Figure III there is a much greater access of first infection in the early school years. About 20% of the population of Sydney never become infected.

These findings have practical application and point to the necessity for certain further inquiries. It was widely believed for many years that by early adult life tuberculous infection was almost universal, a belief based on autopsy studies by Naegeli and others, and by tuberculin surveys carried out in Europe in the early part of the century. This belief has been partly dispelled

by more recent investigations, which have demonstrated wide differences between city and city in the degree of tuberculosis infection of the populations;[6] and the introduction of standards of potency for tuberculin and of standardization of the intracutaneous tuberculin test have made it clear that these differences are not due to differences in technique. In some cities a decline in tuberculosis infection has been demonstrated during the present century; and tendencies for first infections to occur later in life have also been revealed, suggesting that infection is becoming more and more the result of chance contact outside the home as the child grows up, rather than the result of exposure in the home environment in early life.

In Sydney, where the incidence of tuberculous disease is relatively low, the usual age of first infection is late, which supports this idea.

It is still an unsolved problem whether or not it is an advantage for many of the people, as in Sydney, to reach adult life without ever having had occasion to resist the tubercle bacillus. If it is true, as some still believe, that tuberculosis is exclusively a family disease, occurring in those who have received in childhood an infection which has lain dormant and become manifest on the development of favourable circumstances; and that infections in later life are usually milder and more likely to be well resisted on account of the slighter and more transient contacts made outside the home, then the position in Sydney would be reassuring indeed; and, if it could be shown that the average age of first infection was becoming later, diminution of tuberculosis morbidity could be anticipated. This last matter is explored in the next section. But during the past decade it has been shown by Heimbeck[7] and others in Norway that primary infection in early adult life may constitute a real menace to health, even in civilized communities. Heimbeck first observed the health of the subjects of tuberculin tests over a number of years, and found that primary infection in early adult life was often productive of manifest tuberculous disease, while those already infected at the time of first observation seldom or never manifested tuberculous disease. But his subjects were nurses and so were exposed to a greater degree of contact with tuberculous disease; and in Norway tuberculosis is at least three times as prevalent as in Australia.[5] It has yet to be shown that in a population such as ours manifest tuberculosis is other than a rare occurrence in those who sustain infection for the first time in early adult life. This important problem could easily be solved by means of a tuberculin-testing and radiographic survey of university students in medical and other faculties, with observation of the subjects over a period of years. Alternatively, the survey might be made at a large factory or at several large city offices, such as those where the executives have shown themselves sympathetic to the present survey and high proportions of the employees have volunteered for tuberculin tests. Such a survey would be of great benefit to those who participated as subjects, for their health would be checked frequently during that period of life when the onset of tuberculous disease is most common, and many cases of tuberculosis would undoubtedly be diagnosed before severe damage had been done to the lungs.

Very valuable surveys of this kind are already in progress in Scandinavia,[8] but they may be of only limited help to us in our problem, for some of the Scandinavian groups are much more heavily infected with tuberculosis than the population of Sydney, 81% of 2,690 students in one series being reactors to tuberculin at the time of the commencement of their studies. In Sydney the proportion is less than 50%.

The Trend in the Age of First Infection.

As early as 1911 it was stated by Griffiths,[9] who was using Koch's subcutaneous tuberculin test and Calmette's ophthalmic test, that there appeared to be a larger proportion of adult non-reactors to tuberculin in Sydney than in the Old World; he published a number of doubtful cases in which a diagnosis of tuberculosis was excluded with tuberculin. A similar conclusion was reached in 1931 by Harvey,[10] who was using the von Pirquet and Mantoux tests.

The only exact statistics which are available are those published by Penfold[11] in 1923, some of which are reproduced in Tables VI and VII. However, it is likely that the demonstrated incidence of tuberculosis infection was considerably lower than the real incidence, for of 77 patients with undoubted tuberculosis, only 88% reacted to the test, so that "false negative" reactions probably

TABLE VI.

Reactions to the Cutaneous Tuberculin Test of 572 Hospital Patients in Sydney, Melbourne and Adelaide tested in 1923. (Rearranged from *Penfold.*)

Ages.	Males.			Females.		
	Number Tested.	Number of Reactors.	Reactors *per centum.*	Number Tested.	Number of Reactors.	Reactors *per centum.*
0 to 10 ..	5	1	20	2	2	100
11 to 20 ..	96	55	57	61	30	49
21 to 30 ..	110	79	72	96	68	71
31 to 40 ..	103	68	66	99	64	65
Totals ..	314			258		

TABLE VII.

Reactions to the Cutaneous Tuberculin Test of 136 Patients in the Melbourne Children's Hospital tested in 1923 (from Penfold).

Ages.	Number Tested.	Number of Reactors.	Reactors *per centum*.	
0 to 4	8	2	25·0	18·0
5 to 9	70	12	17·1	
10 to 14	58	13	22·4	
Total	136			

resulted in some of the other tests of patients who were not suffering from tuberculosis. In Figure VI Penfold's figures are directly compared with those obtained in the present survey. Even if we disregard the probability that Penfold's figures are too low, a pronounced delay in the age of first infection appears to have taken place during the last fifteen years. The significance of this delay was discussed in the last section.

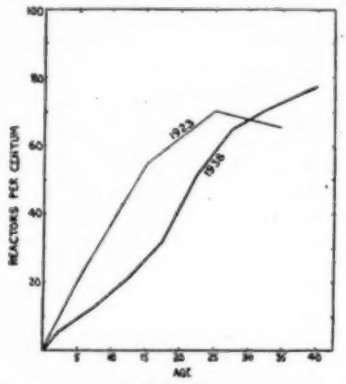

FIGURE VI.

Reactions to the cutaneous tuberculin test of 806 persons tested in Australian cities in 1923 and reported by Penfold (from Tables VI and VII) and the reactions to the intracutaneous tuberculin test of 5,822 persons tested in the present survey.

Comparison of City Dwellers with Country People.

The subjects of this survey were fairly uniform as regards their length of residence in the city. Of 1,254 subjects over the age of fifteen years, 1,132 had lived more than eleven years in the city, and of these, 57·4% were reactors to the tuberculin test; 53 had lived less than four years in the city, and of these, 49·1% were reactors. Owing to the small total number of the latter, they are not here analysed in age groups.

A further survey is being made, embracing large numbers of country dwellers, and will be reported in a separate communication.

Place of Birth and Place of Residence.

In Table VIII (which is illustrated by Figure VII) are set out the reaction to the intracutaneous tuberculin test of 1,326 persons grouped according to their place of birth; but no difference of statistical significance is demonstrated in the degree of tuberculous infection in the various groups. However, a smaller proportion of those born on the north side of Sydney Harbour reacted to the test, age for age, than of those in the other groups.

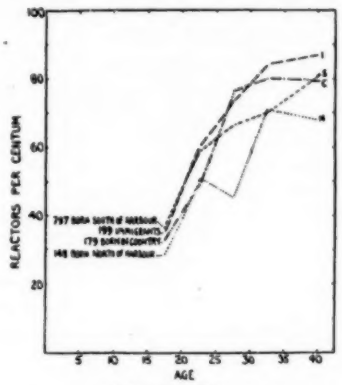

FIGURE VII.

Reactions to the intracutaneous tuberculin test of 1,326 persons grouped according to age and birthplace. (From Table VIII.)

In Table IX are set out the reactions to the intracutaneous tuberculin test of 664 persons grouped according to their place of residence. Again a smaller proportion of those living on the north side of Sydney Harbour reacted to the test, age for age, than of those in the other groups; but some of the age groups were rather small and the differences are probably not of statistical significance.

Occupation.

It is possible to group some 1,466 of the subjects tested according to their occupations, and these

TABLE VIII.

Reactions to the Intracutaneous Tuberculin Test of 1,326 Persons Grouped According to Birthplace.

Ages.	North Side of Harbour.			South Side of Harbour.			Country.			Exogenous.		
	Number Tested.	Number of Reactors.	Reactors *per centum*.	Number Tested.	Number of Reactors.	Reactors *per centum*.	Number Tested.	Number of Reactors.	Reactors *per centum*.	Number Tested.	Number of Reactors.	Reactors *per centum*.
15 to 19 ..	32	9	28·1	265	98	37·0	39	13	33·3	34	12	35·3
20 to 24 ..	59	30	50·8	218	126	57·8	53	26	49·1	42	25	59·5
25 to 29 ..	31	14	45·2	143	95	66·4	39	30	76·9	30	22	73·3
30 to 34 ..	14	10	71·4	67	47	70·1	25	20	80·0	31	26	83·9
35	9	6	66·7	68	58	85·3	19	15	78·9	32	28	87·5
Totals ..	148			797			179			202		

TABLE IX.

Reactions to the Intracutaneous Tuberculin Test of 630 Subjects grouped according to their Place of Residence.

Ages.	South Side of Harbour.						North Side of Harbour.		
	Closely Settled Suburbs.			Less Closely Settled Suburbs.					
	Number Tested.	Number of Reactors.	Reactors *per centum.*	Number Tested.	Number of Reactors.	Reactors *per centum.*	Number Tested.	Number of Reactors.	Reactors *per centum.*
10 to 14 ..	7	3	42·9	4	1	25·0	0	0	—
15 to 19 ..	106	41	38·7 } 43·4	64	36	56·3 } 56·7	30	11	36·7 } 44·1
20 to 24 ..	62	32	51·6 }	70	40	57·1 }	45	22	48·9 }
25 to 29 ..	43	31	22·1 } 60·0	46	28	60·9 } 58·6	24	12	50·0 } 49·3
30 to 34 ..	21	19	90·5 } 78·1	33	26	78·8 } 69·6	7	5	71·4 } 54·8
35 and over ..	17	15	88·2 } 89·8	41	36	87·8 } 83·8	10	9	90·0 } 82·4
Totals ..	256			258			116		

and their reactions are analysed in age groups in Table X. All occupations or quinquennial age groups of occupations comprising less than 20 persons have been omitted. Figure VIII represents Table X graphically.

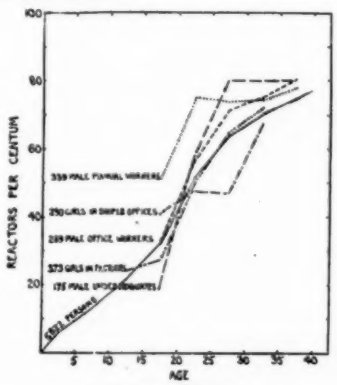

FIGURE VIII.

Reactions to the intracutaneous tuberculin test of 1,466 persons grouped according to age and occupation. (From Table X.)

It is again seen that more men than women are infected, and at an earlier age. Of the men, the manual workers are earlier and more heavily infected than the students and office workers. Outdoor workers are as heavily infected as indoor workers, the "fresh air" availing them nothing. Tuberculin tests have also been applied to more than 1,000 nurses and medical students, and the results will be reported in a separate communication.

Children in Schools, Orphanages and Hospital.

In Table XI are analysed comparatively the reactions of 1,674 children to the intracutaneous tuberculin test. Some were inmates of two large orphanages near Sydney. These children were happy, healthy and very well cared for, enjoying amenities, discipline and upbringing much superior to those of the average child in a working-class home. They were almost a closed community; but the older children went out each day to school in the district, which was semi-rural. Some were in-patients in the Royal North Shore Hospital of Sydney, and came from working-class and middle-class homes on the north side of the harbour. Some were tested at large suburban public schools, all south of the harbour.

Figure IX illustrates Table XI and shows that the orphans and the northern suburbs children had received substantially less tuberculosis infection than the others. Although the numbers are fairly large, the explanation of this is not clear. It is certainly not due to variations in technique, for every test was carried out and read by myself. There was no significant difference between the various schools. A larger survey embracing a large

TABLE X.

Reactions to the Intracutaneous Tuberculin Test of 1,466 Persons grouped according to Occupation.

Ages.	Male Manual Workers.			Male Office Workers.			Male Non-Medical Undergraduates.			Girls in Shops and Offices.			Girls in Factories.			Ages.
	Number Tested.	Number of Reactors.	Reactors *per centum.*	Number Tested.	Number of Reactors.	Reactors *per centum.*	Number Tested.	Number of Reactors.	Reactors *per centum.*	Number Tested.	Number of Reactors.	Reactors *per centum.*	Number Tested.	Number of Reactors.	Reactors *per centum.*	
10–14													33	8	24·2	10–14
15–19	56	29	51·8	69	23	33·3	37	7	18·9	84	35	41·7	183	50	27·3	15–19
20–24	76	57	75·0	93	53	57·0	118	68	57·6	99	47	47·5	95	48	50·5	20–24
25–29	88	65	73·9	66	47	71·2	20	16	80·0	73	34	46·6	40	26	65·0	25–29
30–34	71	53	74·7	20	15	75·0				34	23	67·6	22	16	72·7	30–34
35	48	40	83·3	41	35	85·4										35
Totals	339			289			175			290			373			

TABLE XI.

Reactions to the Intracutaneous Tuberculin Test of 1,674 Children (Orphans, Scholars and Hospital Patients).

Ages.	Orphans.			Scholars (South of Harbour).			Patients (Northern Suburbs).		
	Number Tested.	Number of Reactors.	Reactors *per centum.*	Number Tested.	Number of Reactors.	Reactors *per centum.*	Number Tested.	Number of Reactors.	Reactors *per centum.*
0 to 4	4	0		1	0		94	5	5·3
5 to 9	76	9	10·5	187	34	18·2	177	16	9·0
10 to 14	129	29	22·5	682	146	21·4	110	20	18·2
15 to 19	32	7	21·9	151	45	29·8	31	6	19·3
Totals ..	241			1,021			412		

number of schools in all districts will be necessary to determine the significance of the apparent difference in the incidence of tuberculosis infection in children in various parts of the metropolis.

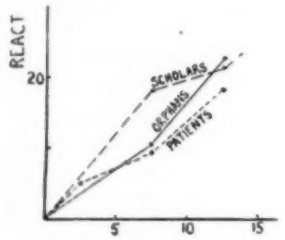

FIGURE IX.

Reactions to the intracutaneous tuberculin test of 1,674 children. (From Table XI.)

Patients in Mental Hospitals.

An interesting series for comparative purposes is provided by 289 patients in a mental hospital near Sydney. The patients, who were selected at random from the more docile of the inmates, are well cared for and live in beautiful surroundings, though of necessity large groups of people live in very close association with one another. Their reactions to the intracutaneous tuberculin test are analysed in age groups in Table XII, which is illustrated by Figure X. The younger patients show a very much higher incidence of tuberculosis infection and a higher rate of infection than the population at large, illustrating well the contagiousness of tuberculosis infection (as distinct from manifest disease). The proportion infected of those over the age of twenty-five is not much greater than in the population at large, though the rate of infection is higher. It is not possible here to correlate the proportions infected and the rate of infection with the length

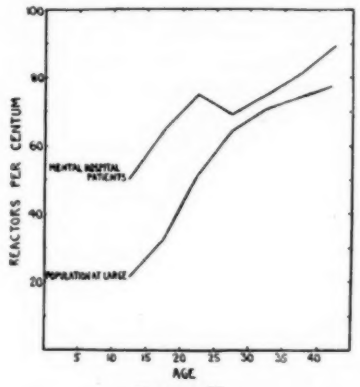

FIGURE X.

Reactions to the intracutaneous tuberculin test of 289 patients in a mental hospital compared with those of the population at large. (From Tables XII and I.)

of time the patients had been resident in the institution; it is sufficient to point out that many of the patients in their late twenties would have been in mental hospitals only a short time, the modes in the age of onset of insanity being a contributory factor to the shape of the curve in Figure X. No evidence is available as to the incidence of manifest tuberculosis in the institution in question. It may here be suggested that the intracutaneous tuberculin test will be a valuable

TABLE XII.

Reactions to the Intracutaneous Tuberculin Test of 289 Inmates of a Mental Hospital.

Ages.	Males.			Females.			Total.		
	Number Tested.	Number of Reactors.	Reactors *per centum.*	Number Tested.	Number of Reactors.	Reactors *per centum.*	Number Tested.	Number of Reactors.	Reactors *per centum.*
10 to 14	1	1	—	1	0	—	2	1	50·0
15 to 19	11	7	63·6	6	4	66·7	17	11	64·7
20 to 24	10	10	100·0	18	11	61·1	28	21	75·0
25 to 29	17	15	88·2	21	11	52·4	38	26	68·4
30 to 34	12	9	75·0	12	9	75·0	24	18	75·0
35 to 39	19	18	94·7	18	12	66·7	37	30	81·1
40 to 44	8	8	100·0	11	9	81·8	19	17	89·5
45 and over	73	62	84·9	51	35	68·3	124	97	78·2
Totals ..	151			138			289		

contributory method in the economical solution of the public health problems occasioned by tuberculous disease in mental hospitals. Indeed the study of tuberculosis infection in such closed communities as these may well shed light into obscure places in the epidemiology of tuberculosis generally.

Summary.

A survey has been made to determine the incidence of tuberculosis infection (as distinct from tuberculous disease) in the population of Sydney. The reactions to the Mantoux test of over 7,000 persons, including 5,822 representing a fair cross-section of the population, are analysed and graphed.

It appears that the average age of primary tuberculosis infection is substantially later in Sydney than in other parts of the world where surveys of the general population have been made, and has become perceptibly later in the last fifteen years. The significance of this is discussed.

It will take some years to determine whether those who reach early adult life without having acquired tuberculosis infection are as specially prone to develop serious disease under local conditions as, according to reports, in other parts of the world. An investigation is suggested which would resolve this question.

A smaller proportion of the population acquires tuberculosis infection in the home in childhood in Sydney than elsewhere, as might be expected in a community with relatively so small an incidence of tuberculous disease. This is not to deny the special liability of members of tuberculous families to develop tuberculous disease, nor the desirability of genealogical investigation in every case of tuberculosis.

At least one-fifth of the urban population remains uninfected by the tubercle bacillus at the age of forty.

The incidence of tuberculosis infection in home contacts is compared with that in non-contacts; it is shown that nearly half the contacts are infected by the age of five years, and nearly two-thirds before puberty.

It is suggested that the incidence of tuberculosis infection is lower in country districts than in Sydney. An investigation to resolve this matter is mentioned.

It is shown that, generally speaking, more men than women acquire tuberculosis infection, and at an earlier age, and some evidence is produced tending to show that infection with tuberculosis varies inversely as the degree of domesticity. Manual workers and office workers by the age of twenty-five to thirty are infected in equal proportions; but, generally speaking, the latter acquire their infection at a much later age than the former.

The incidence of tuberculosis infection in children (scholars, orphans and others) has been studied, and the patients in a large mental hospital have been investigated. Attention is drawn to the value of the tuberculin test in the solution of the public health problems occasioned by tuberculous disease in such closed communities as the mental hospital population.

Acknowledgements.

It is a pleasure to acknowledge the enthusiastic and very great help of Dr. Cotter Harvey, who supervised the work for the National Health and Medical Research Council. I feel much indebted also to the following ladies and gentlemen for helping me in many ways, especially by enabling me to get in touch with the large number of willing and interested people who made the work possible by allowing me to test them with tuberculin: W. P. Ash, Esquire (Honorary Secretary, Sydney University Law Society); Professor E. Ashby; Dr. Keith Barry; A. H. Benning, Esquire (Staff Officer, Australian Gas Light Company); Dr. W. C. M. Dickey (Medical Officer, Australian Mutual Provident Society); M. F. Dixon, Esquire (Federal News Editor, Australian Broadcasting Commission); R. R. Downing, Esquire (Secretary, Textile Workers' Union); the Editors of *The Sydney Morning Herald*, *The Sun*, *Daily Telegraph*, *The Sunday Sun*, *Labor Daily* and *Red Tape*; Dr. G. L. Ewan (Medical Superintendent, Rydalmere Mental Hospital); Miss A. Gardiner (Nursing Sister, David Jones, Limited); Mr. and Mrs. R. E. Hill (Church of England Boys' Home, Carlingford); Dr. John Hughes (Medical Officer, Tuberculosis Division, New South Wales Health Department); Dr. W. W. Ingram; L. L. Isen, Esquire (Director, Clifford Love and Company, Limited); Matron Jenkin (Church of England Girls' Home, Carlingford); E. C. Klindworth, Esquire (Managing Director, Jantzen (Australia) Limited); Lever Brothers, Limited; Dr. A. E. Machin (Principal Medical Officer, New South Wales Education Department); E. D. McNeill, Esquire (Staff Manager, David Jones, Limited); Dr. A. H. Moseley (Medical Officer, General Post Office, Sydney); Sir Norman Nock (Managing Director, Nock and Kirby, Limited); Sir John Peden; L. Philpott, Esquire (Secretary, Students' Representative Council, University of Sydney); J. Plimsoll, Esquire (President, Sydney University Evening Students' Association); the Professorial Board, University of Sydney; J. W. Powell, Esquire (Managing Director, Foster Clark (Australia) Limited); Dr. J. R. Radcliff (Medical Superintendent, Royal North Shore Hospital of Sydney); Dr. Clarence Read; T. W. Ritchie, Esquire (Compensation Department Officer, Trades and Labour Council of New South Wales); S. Rutherford, Esquire (Secretary, Colgate Palmolive, Limited); Dr. George Saunders (Medical Officer, Metropolitan Water, Sewerage and Drainage Board); A. Tyler, Esquire (General Manager in Australia, Parke, Davis and Company); T. H. Upton, Esquire (President, Metropolitan Water, Sewerage and Drainage Board); Dr. H. G. Wallace; R. H. Woodbridge, Esquire (General Manager, Australian Gas Light Company).

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SOME ASPECTS OF THYREOTOXICOSIS IN NEW SOUTH WALES.[1]

By NORMAN WYNDHAM,
Sydney.

Incidence.

THE incidence of thyreotoxicosis in New South Wales is apparently increasing. While it is not in any way conclusive, it is worth noting that the number of patients treated at the Royal Prince Alfred Hospital, Sydney, has become greater during the ten-year period from July, 1927, to July, 1937. The figures are shown in Table I.

TABLE I.

Period.	Number of Cases.		
	Surgical.	Medical.	Total.
July, 1927, to June, 1928	71	33	104
July, 1928, to June, 1929	72	28	100
July, 1929, to June, 1930	71	23	94
July, 1930, to June, 1931	64	25	89
July, 1931, to June, 1932	81	18	99
July, 1932, to June, 1933	90	37	127
July, 1933, to June, 1934	88	48	136
July, 1934, to June, 1935	84	69	153
July, 1935, to June, 1936	102	73	175
July, 1936, to June, 1937	104	46	150

There was no increase in bed capacity at the hospital during this period, but from 1930 to 1932 there were fewer beds. Of more significance, however, is the rising death rate, not only in this State, but in the Commonwealth also. This increase is found in Great Britain. (See Figures I and II.)

It is important to consider the problem from the therapeutic, ætiological and preventive points of view. Since the introduction of iodine therapy as a pre-operative measure, the standardization of operative technique and improved anæsthesia, we have arrived at an apparently irreducible minimum of some 1% mortality in the hands of surgeons who treat many patients. The mortality from medical treatment is difficult to assess, since many failures are handed over to the surgeons. Of course, some may hold that the converse is correct. It is also unfair to criticize the results from irradiation, since this method is reserved in most clinics for patients whom the surgeon is not anxious to touch. It is felt, however, that the benefit to suffering humanity from the introduction of iodine therapy has been largely counterbalanced by its injudicious and prolonged use in the treatment of some patients suffering from thyreotoxicosis.

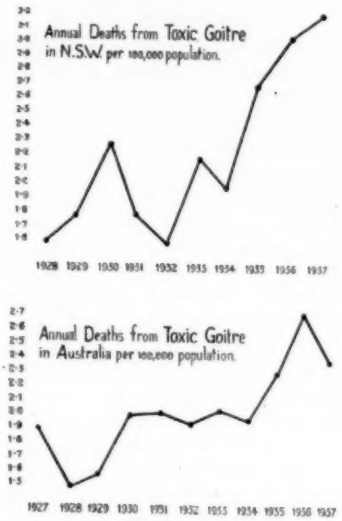


FIGURE I.

Geographic Distribution.

An investigation was made of 887 patients with thyreotoxicosis who had been treated by various physicians and surgeons, with reference to the part of the State from which they came. The numbers of years spent at each place and the time of onset of signs or symptoms were noted. The incidence was twice as high in the city as in the country areas. In the figures of a city hospital this is of no significance. An endeavour was made to prepare a map showing the percentage incidence of the disease in the State outside the County of Cumberland, on the basis of total population of shires and municipalities. (See Figures III and IV.) As it was known that toxic goitre is treated in various places in the country areas, additional information was obtained from practitioners in these localities. The relative incidence in city and country is roughly that found in other

FIGURE II.

[1] Work done with the aid of a grant from the National Health and Medical Research Council.

parts of the world. The highest incidence throughout the State, if Sydney and Newcastle are left aside, is in the region of the watersheds. There appears to be a close correlation between the incidence of toxic and endemic goitre. There is agreement on this point in other countries. A summary of the accepted ideas of the Third International Goitre Conference is given by Eggenberger, in a paper entitled *"Jodthyreotoxicosen und ihre Verbütung durch biologische Joddosen"*.

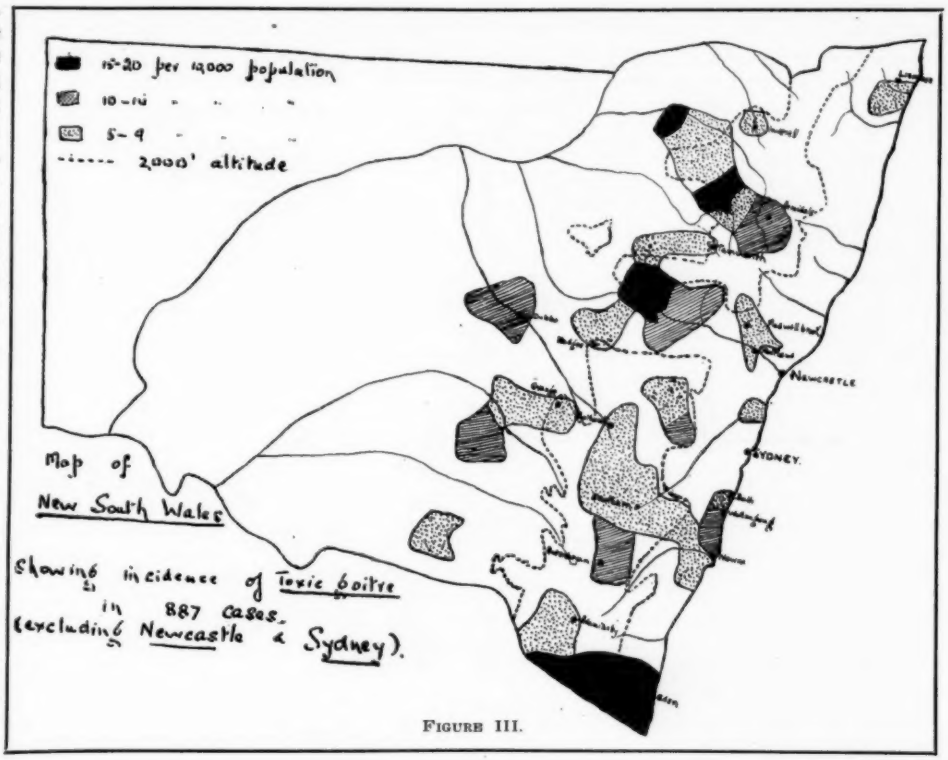


FIGURE III.

The relative incidence in city and country varies according to classification of the type of goitre. A rough classification was made in 381 cases by Dr. H. R. G. Poate, based on history, physical examination and pathological

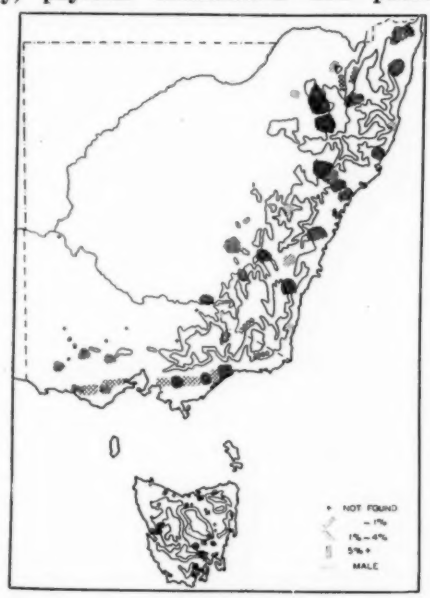


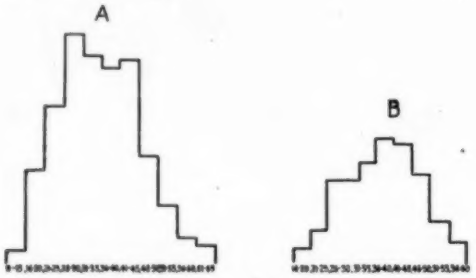
FIGURE IV.
Goitre percentage incidence in school children in association with contours of 2,000 and 3,000 feet in eastern Australia. (Reproduced from an article by Harvey Sutton in *The Australian Geographer*, Volume II, Number 2.)

examination. By these criteria, 270 cases were of so-called "primary" toxic goitre; in 111 there was a history of a previous non-toxic goitre which was supported by the histological appearance. In the "primary" cases 185, or 69%, of the patients came from the city. In the "secondary" cases, 50% of the patients developed symptoms while living in the country, and most of the others had lived in the country during the earlier part of their life.

Relation of Thyreotoxicosis to Endemic Goitre.

In view of the seriousness of the toxic goitre problem in this country, it is important to realize that in roughly one-third of cases the underlying ætiological factor was an endemic or non-toxic goitre in early life. In other words, it is probable that most, if not all, of the one hundred and eleven cases in this group could have been prevented by the correct preventive treatment of endemic goitre. Some will deny this and contend that the non-toxic goitre was incidental and that thyreotoxicosis would have developed irrespective of this condition. The belief of many who have watched the development of these cases is that this endemic goitre of early life is very likely to develop into a more serious condition in later life. The position in this country may be different from that which obtains elsewhere; but I believe that it is a rarity for a person with any kind of goitre to pass through life free from symptoms of some sort or another referable to the thyreoid gland. In many instances it is a frank thyreotoxicosis. The age at which this occurs is variable. Graphs of the age incidence in primary and secondary toxic goitre show that

the greatest number of cases do not occur in the same decade in each type. (See Figure V.) Young women between twenty and thirty years are the most frequent sufferers from thyreotoxicosis of "primary" type, while the other cases are most often found in the fifth decade. This is the universal finding.



Graphs showing peaks of incidence of toxic goitre in 5 year age periods.
A. primary toxic goitre. B. secondary toxic goitre.

FIGURE V.

There seems to be, therefore, a natural tendency for these non-toxic goitres to become hyperplastic and hyperfunctional in middle life. In some cases the exhibition of iodine seems to be the requisite stimulus to this change. Carmalt Jones (1928) made the following statement:

> Such goitres may be treated with iodine; in children, re-adjustment to the increased intake is readily made, but in adults long accustomed to a low intake, excess often causes too great hormone production, with toxic symptoms.

While one may not agree with this statement as to the mechanism of the establishment of thyreotoxicosis, it should be remembered that Lugol's iodine solution as usually administered contains a huge quantity of iodine compared with the normal body requirements. The following is but one of many instances of a similar nature which have come to my notice, and shows that even young persons may be the victims:

> K.M.W. was a single girl, aged seventeen years, who came from an "endemic" area; both her parents had non-toxic goitre. At the age of fifteen years she consulted her own doctor for a symptomless, diffuse colloid enlargement of the thyreoid gland, present since puberty. This was treated by a method widely recommended in other countries, namely, the administration of two minims of Lugal's solution twice a day and half a grain of dried thyreoid gland twice a day. She had instructions to return for examination in three months' time, but failed to do so until nineteen months had elapsed, when she was seen by Dr. Poate. At this time she complained of not feeling well and had all the signs and symptoms of severe thyreotoxicosis. She was obese, owing to a voracious appetite; she had exophthalmos; her initial basal metabolic rate was +63%.

"Iod-Basedowism", a well-known entity in the early part of this century, and a condition which postponed the use of iodine in toxic goitre for some years, has apparently been forgotten in this country.

It must be admitted that no such chain of events has been apparent in the histories of the majority of our patients with secondary toxic goitre. It appears that a non-toxic goitre can be converted into a toxic goitre by the stimulation of those stresses and strains in life, mental or physiological, which leave the normal person unaffected.

It is obvious in the study of this problem that a much larger proportion of the cases occurring in this State are of the secondary type, compared with the statistics of other countries. The reason for this is obscure; but it is felt that many writers abroad are inclined to leave these cases out of their discussions. They do not fit conveniently into theories of ætiology, pathological anatomy and biochemistry, or of treatment. I cannot, however, ignore 111 or a third of the cases collected by Dr. Poate and myself, nor should it be forgotten that it seems more than likely that these cases could have been prevented by adequate measures directed towards the elimination of endemic goitre. It is not the purpose of this paper to discuss this preventive management. It can be pointed out that successful efforts have been made in other parts of the world—for example, in North America (Marine, 1935), Switzerland (Eggenberger, 1926), New Zealand (Tubiasz, 1938), Bavaria (Fischler, 1934). Whether or not other causes act in the development of endemic goitre as a suggested by Spence (1934) and McCarrison (1935), most seem to agree that "iodine acts as a shield against endemic goitre and iodine prophylaxis is the most important preventive measure". This does not mean the administration of massive doses in the form of Lugol's solution.

The terms "primary" and "secondary" have been frequently used in this paper, and have probably given the impression that this classification is easy and obvious. This has not been my experience. The exact nature of the thyreoid gland is frequently difficult to determine before operation, and most pathologists will admit that it is often impossible to tell whether one is looking at a section of a colloid-filled gland which has become hyperplastic or a hyperplastic gland which is reacting to iodine therapy. In the absence of definite nodular areas one must often rely on the history given by the patient as to the preexistence of a goitre. How unreliable this is one realizes when one considers the number of patients who did not know that they had a goitre until told by their doctor, and further when one thinks of the number of patients with a toxic goitre which has escaped the notice of the doctor himself.

How many of these diffuse, hyperplastic glands may have been simply enlarged originally? This previous goitre may have been physiological. For example, in 8·8% of the "primary" cases in this series the onset of symptoms was associated with parturition. A physiological enlargement during pregnancy may subside, to become hyperplastic and hyperfunctional at a later date, usually after a subsequent pregnancy. The onset of toxic symptoms occurs usually in the third month of lactation. What is the proper classification of these cases? They would point to the wisdom of the use of iodized salt during prenatal treatment.

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TICK PARALYSIS: A DANGEROUS DISEASE IN CHILDREN.

By D. G. HAMILTON, M.B., B.S., M.R.A.C.P.,
Medical Registrar, Royal Alexandra Hospital for Children, Sydney.

IT is known to most Australians that tick paralysis is a dangerous disease in dogs. It is realized by far too few, however, that it can affect and prove fatal in human beings, particularly children. The disease is known to occur in four continents: Australia, North America, Africa and Europe, where four different species of tick are regarded as the causal agents, namely, *Ixodes holocyclus* (in Australia), *Dermacentor venustus, Ixodes pilosus,* and *Ixodes ricinus.* In addition to dogs and humans, cats, pigs, fowls, calves, foals and sheep are known to have been affected.[1] All native fauna are probably immune.[2]

The disease consists of a widespread lower motor neurone paralysis, which commences after the tick has been feeding for some days, and gradually increases in severity. The onset of the weakness is commonly in the legs and may be preceded by paræsthesia. The child is noticed to be unsteady when walking, the gait at first being ataxic rather than weak. Soon weakness is apparent. He staggers a few steps, and his legs give way under him. Having fallen, he has difficulty in rising. Within the next day or two this weakness increases and spreads. Soon the child cannot walk at all. The arms become tremulous on effort, and soon obviously weak. The trunk and neck muscles become limp, and the child has difficulty in sitting up and holding up his head. Because of the flabby abdominal muscles constipation may be severe.

The muscles of the face may be affected. The cheeks are then smooth, the mouth sags open and the face is expressionless. Involvement of the external eye muscles produces a transient squint or diplopia if muscle imbalance exists. Usually, however, there is no squint, but if the eyes are turned to one side, nystagmus appears, for the weakened muscles are unable to hold the eyeball steady in this strained position. It is akin to the unsteadiness of the gait and the tremor of the arms on exertion before frank muscle weakness is apparent. Weakness of the intrinsic eye muscles produces photophobia or blurring of near vision. All too frequently the muscles of deglutition are involved and the child cannot swallow. Food regurgitates from the limp mouth, fluids perhaps through the nose. The voice becomes thick and indistinct, but not nasal unless the palatal movement is impaired. If the muscles of respiration are involved the respiratory rate becomes increased, the breathing laboured and the child restless and anxious. The *alæ nasi* become dilated. Cyanosis may develop. The voice and cough become feeble. The expansion of the chest from intercostal action is noticeably lessened, or the bulge of the epigastrium from the downward movement of the diaphragm during inspiration disappears. As the child's restlessness increases, the pulse becomes increasingly rapid and feeble. The temperature frequently rises several degrees, but the patient may remain apyrexial throughout the disease.

The height of the weakness is reached approximately forty-eight hours after the tick drops off or is removed from the body. If death occurs it is from respiratory failure, or later from aspiration pneumonia caused by careless feeding at the height of the paralysis. Once this period of maximum paralysis is passed, a gradual recovery sets in and becomes complete in every respect after a variable period. No residual paralysis is left.

Neurological examination reveals evidence of a widespread paralysis of lower motor neurone type. The tendon reflexes are absent, and the abdominal reflexes are often diminished. The plantar response remains flexor in type. The reaction of the pupil to light and accommodation may be diminished, but seldom very greatly. Nystagmus may be elicited. Muscle wasting is but slight, for the disease is of short duration. Objective sensory changes are not found. The cerebro-spinal fluid, in the one case in which it has been examined at the hospital, was normal.

The weakness is not always so severe and so widespread as has been described above. As with dogs, the natural immunity of human beings to tick venom probably varies. The size of the patient and the amount of venom injected also determine the severity of the disease; and the amount of venom depends on the tick itself as well as on its degree of engorgement.

In some cases a purely local paralysis develops. Thus a bite on the face or in the ear may cause nothing but a facial paralysis. In one of the hospital cases described below, a bite on the forehead caused paralysis of the frontalis and *orbicularis oculi* muscles alone.

Australian medical literature contains reports of a number of cases of both general and local types.

In 1924 Ferguson[3] reviewed the fatal cases which he was able to trace from published reports or from the Government Statistician's records. He describes the following cases of fatal generalized paralysis:

1. A girl, aged thirteen months, died at Eden, on the south coast of New South Wales, in 1912, death being due to respiratory paralysis. The tick was attached behind the left ear.

2. A girl, aged three years, from Wagonga, died in 1913; the tick was attached to the chest, almost in the axilla.

3. A child aged thirteen years, from Mullumbimby, on the north coast of New South Wales, died from respiratory paralysis. The tick was attached in the auditory meatus.

4. A patient from Bellingen, on the north coast of New South Wales, died in 1910. The tick was attached to the forehead.

5. A boy, aged two years, died at Gosford, on the north coast of New South Wales, in 1916.

6. A boy, aged ten months, died at Matcham, near Gosford, in 1921, the tick being attached to the neck.

7. A boy, aged three years, died at Eastwood, a suburb of Sydney, in 1921. In this case there was alleged to be no paralysis of the muscles, and some days were said to have passed between the removal of the tick and death. Heart failure from tick poisoning was given as the cause of death.

8. A boy, aged sixteen months, from Auburn, a suburb of Sydney, died from generalized paralysis from a tick bite in the scalp. Autopsy revealed in all parts of the brain "intense engorgement of the vessels with the presence of numerous small newly formed capillaries; in places diffuse infiltration with small round cells, apparently plasma cells, was noted; there were, however, no perivascular sheaths or cuffs of small round cells. In the spinal cord similar congestion was present with small hæmorrhages".

Moss,[4] in 1924, described a case which occurred in a northern suburb of Sydney.

The patient was a woman, aged thirty-five years. The tick was attached to the inner side of the right *labium majus*, and was alleged to have been there for ten days. Pain and weakness had been present in the legs for five days, and blurring of near vision for two days. Recovery occurred in two days after removal of the tick. The tick was identified as *Ixodes holocyclus*.[5]

In 1930, Sinclair[6] reported a fatal case from Longueville, Sydney, the victim being a child aged three and a half years. The tick was removed, probably on the fourth day of engorgement. Generalized paralysis developed within the next twenty-four hours, and the child died suddenly about forty hours after the tick was removed.

A case reported in 1931 by Foster[7] from Melbourne described a facial paralysis caused by a tick in the auditory canal of the same side in a man, aged forty-five years. The patient recovered three days after removal of the tick.

The facial paralysis caused by a tick in a similar site in a case reported by Crossle,[8] in 1932, however, persisted for three weeks. The patient was a child, aged two years, from Bulli, on the south coast of New South Wales.

Cleland[9] describes intense itching with some prostration lasting several days as the result of the attachment of several hundred larvæ to an adult. He implies, but does not state, that the species was *Ixodes holocyclus*.

The only tick that has been proved to cause paralysis in Australia is *Ixodes holocyclus*,[2] which is called variously the dog tick and the bottle tick. It is sometimes wrongly called the scrub tick and the cattle tick. It has small brown legs and head and a larger rounded greyish body, which becomes darker and bluish grey as it distends with blood. On the ventral surface of the tick's body is the anus, and in relation to the anus a groove. In most ticks this groove is placed behind the anus, but in the genus *Ixodes* the groove surrounds the anal aperture, and in the poisonous species *Ixodes holocyclus* it is closed behind the anus. The groove is readily seen with a good magnifying lens, and the tick may thus be identified. (See Figures I, II and III.)

This particular tick occurs only in a narrow coastal belt a few miles wide extending along the

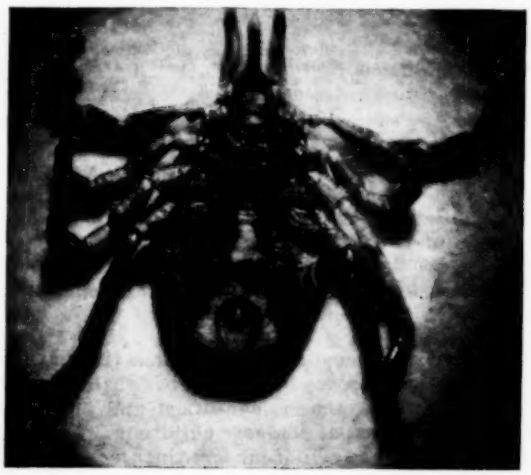

FIGURE I.
Female *Ixodes holocyclus* before feeding. Ventral aspect. The position of the anal groove can be seen. × 14.

east coast of New South Wales and Queensland, and in a similar limited coastal belt in Western Australia. For its survival the parasite must have

FIGURE II.
Proboscis of female *Ixodes holocyclus*. The proboscis of the male is much shorter. × 35.

ample rainfall and a warm temperature. It depends, too, on the presence of bandicoots, which act as host to both immature and mature forms of the tick. They are always heavily infested and are an important factor in the survival of the tick in any district.

All these criteria are satisfied in the coastal regions mentioned above. The danger becomes very real in the suburbs of Sydney in the late spring, and in the summer and autumn months.

The tick that causes paralysis is always the mature female. For a reason that is not understood, however, many mature females of the species *Ixodes holocyclus* are non-toxic.[1] Picked up by the human from scrub, from greenery or flowers in a house or from another animal, the tick attaches itself to any part of the body and feeds for a variable

FIGURE III.
The anal groove of *Ixodes holocyclus*, surrounding and closed behind the anus. ×108.

number of days. When fully engorged it drops off, lays several thousand eggs and dies. The stages of development from the egg are larva, nymph, adult. The period of engorgement of the adult is usually five to seven days, but has been known to vary from four to fourteen days. Engorgement is at first slow, but after several days becomes very much more rapid. Ross has found that an unfed female tick weighs approximately one milligramme. On the fourth day it may weigh 20 milligrammes, but on the sixth day as much as 450 milligrammes.[1] (See Figures IV and V.)

There is good evidence that the venom is produced in the salivary gland of the tick. During the early days of engorgement this gland is small, but during the final days of rapid engorgement it increases tremendously in size. During these final days paralysis commences, and increases for one or two days after the tick drops off or is removed. The toxin passes from the salivary glands into the foremost part of the tick's alimentary tract, and thence into the body of the host, some workers think by active injection, but most believe that it is introduced passively by the pressure of the body rapidly distending behind. A small amount of toxin has been found in the tick's salivary gland in the earlier days of engorgement, and it may be possible for paralysis to occur in a small animal in the first two or three days of attachment, especially if infestation is multiple.

FIGURE IV.
Mature female *Ixodes holocyclus* before feeding. ×6.

A protective serum has been prepared from immune dogs heavily infested with ticks, and is of value in treating poisoned dogs and probably humans.

Treatment.

The first and most important step in treatment is to remove the tick without the least delay, and to examine the patient's body carefully for evidence of multiple infestation. A tick can readily become concealed in the crevices of the body—the auditory meatus, the natal cleft, the vulva or even the nose

FIGURE V.
Mature female *Ixodes holocyclus* fully engorged. Compare with Figure IV. The body has enlarged tremendously. Comparison of the size of the legs will show how closely the ticks corresponded before the one engorged. ×6.

or mouth of a small child. All these parts should be carefully inspected. Removal of the tick is not always easy because of the barbed hypostome by which it is attached. During the early days of engorgement it is safe enough to kill the tick with ether, turpentine or some such substance and

remove it with forceps. Should the hypostome break and remain in the patient it can safely be left there. When the tick is engorged, however, the salivary glands are large and contain an appreciable amount of toxin. The pressure of forceps may squeeze enough of this into the patient to increase the paralysis; therefore, if the tick remains attached after being killed with ether, it should be excised without being squeezed. A very tiny piece of skin is all that need be removed, and fine scissors are the most convenient instrument. If these are not available a useful trick is to loop a strong thread round the tick's head and pull.

The anti-tick serum prepared by the Commonwealth Serum Laboratories from immune dogs is probably of real value in neutralizing toxin not yet fixed in the nervous system. The dose is 10 to 20 cubic centimetres by intramuscular injection and may be repeated if desired. The initial dose, however, is the one most likely to be effective. Whether the serum has any effect on the toxin once it has reached the spinal cord and caused paralysis is not known. Unfortunately, this serum is difficult to obtain in Sydney.

It has become almost habit with many people these days to prescribe large doses of vitamin *B* (especially B_1) for any ill-understood flaccid paralysis. While not greatly impressed by its value, I admit that it is unlikely to do harm and may possibly do some good.

All other treatment is symptomatic. If deglutition is difficult, feeding by a tube passed through the nose into the stomach is imperative. If mucus, saliva and food débris collect in the pharynx and can be neither swallowed nor expelled, the head of the bed should be lowered and the pharynx cleared of accumulations at intervals with a Yankauer's sucker. Atropine given by hypodermic injection, or added to the feedings, checks the flow of saliva and mucus.[10]

Respiratory failure must be treated by continued artificial respiration, only possible in a mechanical respirator.[10] The indications for treatment in the respirator of a patient with weakness of the respiratory muscles are (i) increasing restlessness, (ii) loss of voice and cough, (iii) approaching exhaustion, as shown by sweating and increasing pulse and respiration rates.

Is is usually wise not to wait until cyanosis develops. It should be remembered, however, that the use of the respirator is not without risk. If the child fails to synchronize his breathing with the machine, the experience is an alarming and exhausting one, and may jeopardize the child's life. A baby it may kill quite rapidly. Therefore, unless a child is old enough to understand and obey the simple instruction "Breathe in, breathe out", it is wise not to use the respirator unless its use becomes imperative.

As recovery commences, active movement, massage and hydrotherapy are of some value, though usually not necessary. Splinting is necessary if bad positions, for example, foot-drop, are being assumed. This is the only deformity that is likely to cause trouble, for recovery becomes complete within a few weeks or even days of removal of the tick.

Case Reports.

During the last year four patients with tick paralysis have been treated at the Royal Alexandra Hospital for Children, and these, with three further cases from the hospital records of an earlier period, illustrate the characteristic features and a number of the variations of the disease.

CASE I.—A.R., a girl, aged two months, was a healthy breast-fed baby who lived with her parents and two elder children in a flat in the heart of Sydney. There were no garden and no domestic animals, and the baby had not been taken to a park for a period of seven days preceding the discovery of the tick in her right breast. Apparently the tick had been brought into the flat by one of the older members of the family on the clothes or on flowers or greenery. Unfortunately the mother was advised to leave the tick *in situ*, "as it would come out of its own accord in a few days". Almost three days later the mother brought the child to the hospital. The tick was then engorged to a degree suggesting four to five days' attachment; the child was severely paralysed in the legs and arms, and looked extremely ill. She had difficulty in swallowing and her cry had become very feeble. The knee jerks and abdominal reflexes could not be elicited. The temperature was 99° F. and the respiratory rate 28 per minute. The pulse rate was 130 per minute and the pulse volume was poor.

The tick was immediately removed and the child was given 10 cubic centimetres of canine anti-tick serum by intramuscular injection. She was fed by a tube passed into the stomach. Next morning the child was much worse. The temperature had risen to 103° F. and the respiratory rate to 40 per minute. There were no signs suggestive of pneumonia. During the morning the child became progressively more distressed. The pulse and respiratory rates increased. Cyanosis developed and was not relieved by the administration of oxygen. A fatal termination seemed certain. With some trepidation, therefore, the child was put into the respirator, and she died immediately. The machine was stopped at once, but both respiration and circulation had failed.

There is little doubt that use of the respirator accelerated a death that would have been not long delayed. The tick was identified as a female *Ixodes holocyclus*, from half to two-thirds replete.

CASE II.—J.K., a girl, aged one year and nine months, lived at North Ryde, a suburb of Sydney. On October 22, 1939, the parents noticed that the child's gait was unsteady and that her left leg occasionally gave way under her. On October 23 the child brought and gave to her mother a tick. From the description given by the parents the tick was approaching or had reached repletion. The child's gait was now more unsteady and the right leg also was weak. The child would walk a few steps unsteadily and her legs would then go from under her. Having fallen, she had difficulty in rising. There was, however, little demonstrable muscle weakness on physical examination.

On October 24 the child was admitted to hospital. She was then unable to walk at all. Examination revealed a flaccid weakness of both legs. The child could use all her muscles, but feebly. The knee and ankle jerks were absent and the abdominal reflexes were diminished. A small puncture wound surmounting an indurated base showed the site of attachment of the tick in the left occipital region. On October 25 the child's condition was still worse. The weakness was more pronounced in the legs and the abdominal muscles, arms, neck and face were now slightly involved. Lumbar puncture under local anæsthesia revealed clear fluid containing no cells and containing 20 milligrammes of total protein and 770 milligrammes of total chloride per 100 cubic centimetres.

Sugar was shown to be present by Benedict's reaction and globulin absent by Pandy's reaction. The fluid was sterile on incubation and gave no response to the Wassermann test.

On October 26 the child's condition was still worse. The respiratory muscles, both those of the diaphragm and the intercostals, were slightly involved, and the child was beginning to show signs of restlessness and distress. She was given 10 cubic centimetres of canine anti-tick serum by intramuscular injection, and alkalis and 333 new international units of vitamin B_1 three times a day were exhibited. At 12.30 p.m. the child was put into the respirator. Synchronization was satisfactory. The same day paralysis of the muscles of deglutition became apparent. Tube feeding was therefore instituted, atropine was exhibited, the respirator was tilted and the pharynx was cleared of accumulated saliva with the sucker at intervals. On October 27 the child's condition was satisfactory, but unchanged. On October 28, six days after the onset of weakness, improvement commenced, and within a further twenty-four hours the child was able to eat and breathe normally and to move her limbs without difficulty. Unfortunately she developed scarlet fever, but this did not retard the steady return of muscle power, and she was discharged from hospital, well, after five weeks.

It is impossible to say how long a time was necessary for complete recovery of muscle power, for the child's period in bed was prolonged by the scarlet fever.

CASE III.—I.W., a boy, aged seven years, lived at Eastwood, a northern suburb of Sydney. On November 6, 1938, the boy complained of photophobia soon after waking, and screwed up his eyes. About midday a tick was found behind the boy's ear and removed. By this time his gait was unsteady. He "staggered like a drunken man". Four hours later he complained of double vision. On November 7, at breakfast, he had difficulty in feeding himself. He managed better at lunch, but at the evening meal he could not swallow and showed signs of choking on attempting to do so. Soon afterwards his breathing was noticed to be distressed and he was sent to hospital.

Physical examination revealed a rather pale, nervous type of boy, lying restlessly, making movements that were almost choreiform in type. There was widespread impairment of motor power. He could move his limbs in all directions, but was quite unable to support his own weight on his legs or to perform steady controlled movements with his arms. He could sit up, but was inclined to sway and fall. The abdominal reflexes were absent. The left knee jerk was absent and the right diminished. Muscle coordination was poor and ataxia appeared to be present in excess of that caused by the weakness. A coarse tremor of the limbs was present on active movement—for example, when the arms were held extended. No impairment of sensation was elicited. Photophobia was present. The right pupil was larger than the left, but both responded, though somewhat sluggishly, to light. Diplopia was sometimes present, but vision was otherwise good. A slight squint was present. The eyeballs could be moved freely in all directions, but if they were held looking to one side an obvious nystagmus developed, the quick component being in the direction of the deviation of the eyes. The muscles of the right side of the face were slightly weak. The palate moved evenly but sluggishly. The pharyngeal muscles were weak, as shown by difficulty in swallowing, by a thick, indistinct, but not nasal quality of the voice and by the presence of an excess of saliva and mucus in the throat. The voice and cough were weak, but there was no respiratory distress. The temperature was 99·6° F. and remained between 99° and 100° F. for ten days. The scar of the tick bite was a small indurated lump surmounted by a dried scab on the occipital region.

The boy was given 10 cubic centimetres of canine anti-tick serum. On November 8 the photophobia, tremor and nystagmus were much less and the abdominal reflexes were elicited. A further 10 cubic centimetres of anti-tick serum were given. On November 9 an improvement in all muscles was obvious. The knee jerks were elicited. Swallowing was confident and controlled, but the voice was still indistinct. The squint was still present.

A steady improvement continued from day to day. Ten days after the first dose of serum a severe urticaria developed. Five weeks after the child's admission to hospital recovery was almost complete. He was walking, though a little unsteadily, and he was discharged from hospital. He soon became normal but for a tendency to tire easily. This passed and he is now quite well. The tick was brought to us by the parents and identified as a female *Ixodes holocyclus*, about two-thirds replete.

CASE IV.—D.R., a girl, aged seven years, living at Ashfield, Sydney, was admitted to hospital on December 6, 1938. Eight days previously she was alleged to have become feverish and a small lump was felt in the scalp above the right temple. She had a cough and was put to bed, where she remained for a week. At the end of this period she had recovered from the cough and fever, but for the first time complained of pain in the right side of her head, and was unable to raise the right eyebrow. The parents therefore examined the lump on the scalp again, found it was a tick, removed it and brought the child to hospital.

Examination revealed paralysis of the right frontalis and *orbicularis oculi* muscles, with ptosis of the upper eyelid and eyebrow. There was no weakness or alteration of reflexes in the other parts of the face, trunk or limbs. Muscle coordination was good and the gait normal. There was no loss of sensation anywhere. There was a puncture wound in the scalp above the right temple, which was slightly inflamed and tender and surrounded by a limited area of œdema. The right posterior cervical and post-auricular lymph glands were slightly enlarged and tender.

The child remained in hospital for fourteen days. During this time little improvement in the paralysis occurred and she was discharged. The paralysis improved very slowly and recovery was finally complete about three months later.

Unfortunately, the tick was not brought for identification.

CASE V.—E.B., a boy, aged ten years, living at Oatley, a suburb of Sydney, was referred to the hospital and admitted on November 25, 1932. Twelve days previously he had complained of pain in the calves of the legs, but continued to attend school. Three days later his legs gave way under him in the street and he was carried home. There he remained in bed for nine days until admitted to hospital; during this period there had been no symptoms except weakness of the legs.

On his admission to hospital a large tick was removed from the scalp, but was not identified. The very meagre clinical notes available record that there was paresis of all muscles of the legs, particularly the dorsiflexors of the feet. The knee jerks were sluggish and the plantar reflexes flexor in type. The child recovered rapidly from the weakness and was discharged from hospital, well, thirteen days later.

CASE VI.—A.T., a girl, aged five years and nine months, was admitted to hospital on December 11, 1927. She lived at Strathfield, but eight days before her admission she had attended a picnic at Clifton Gardens, on the shores of Sydney Harbour, where a tick became attached to another member of the party, but was removed. The patient complained of a lump on her head, but it was not recognized by the parents as a tick and was not removed. The day before her admission to hospital, that is, seven days after the suspected day of attachment of the tick, the child became unsteady on her feet. The weakness progressed rapidly. On her admission to hospital pronounced flaccid paralysis of the legs and arms was present. The knee jerks were absent. Cyanosis and sighing respiration indicated involvement of the respiratory muscles. The note "salivating profusely" probably refers to the accumulation of saliva and mucus in the mouth and pharynx, and indicates paralysis of the muscles of deglutition. Nystagmus was present, but the pupils reacted to light. The child was semi-comatose. A tick,

described as "about the size of an almond", was removed from the scalp and identified as *Ixodes holocyclus*. The temperature rose to 100° F. and the child died a few hours after her admission to hospital.

CASE VII.—N.McC., a boy, aged ten years, living at Surry Hills, in the city of Sydney, was brought to hospital on December 30, 1924, complaining of weakness of the right arm of three days' duration. Approximately ten days before the onset of weakness the boy noticed a "wart" in the right axilla. Examination revealed severe paralysis of the dorsiflexors of the wrist and paresis of the other muscles of the arm and forearm. The "wart" was found to be a tick. Unfortunately the degree of engorgement is not noted, nor was the species of tick ascertained. The tick was removed and in six days power was completely restored in the arm.

Table I gives a summary of the cases reported.

Comment.

It is plain that tick paralysis is an important disease and should be thought of whenever a human being, especially a child, develops an acute lower motor neurone paralysis, whether generalized or localized. The whole body should be carefully searched for ticks, particularly the scalp and the body crevices. Fortunately the disease is localized geographically.

In general, the younger the child and the greater the engorgement of the tick, the more severe is the paralysis.

The cases reported illustrate many of the variations of the disease, some of the sites of attachment of the tick, the variable rate of engorgement, the variable site, degree and duration of paralysis, and the progress of the paralysis after removal of the tick.

It is interesting to speculate as to why paralysis is sometimes generalized and sometimes localized to the region of the bite. McKay[11] suggests that localized paralysis is due to a local œdema, probably allergic in type, causing pressure on the regional nerves. In this respect it is interesting to note that many people have become sensitive to the bites of the tick and develop urticaria whenever bitten. This, however, is widespread, and appears within an hour or so of attachment of the tick. Ross himself has suffered in this way.

A more likely explanation is that the tick venom is neurotropic and is absorbed by the peripheral nerves, usually travelling along them to the central nervous system. In a proportion of patients, however, some degree of tissue sensitivity or tissue resistance in these nerves causes the toxin to become fixed there and temporarily paralyse the nerve. This local fixation of toxin may be in the peripheral nerve itself or in the corresponding segment of the spinal cord. Wherever it is, it is motor function alone that is paralysed.

Another possible explanation is that local paralysis is caused by a different species of tick. As far as I have been able to find, the tick causing such paralysis has never been identified.

All these issues should be readily solved by careful observation and investigation.

The site of action of the toxin in generalized paralysis seems to be the lower motor neurone of the spinal cord and cranial nerves. There is no clinical evidence of involvement of the upper motor neurone. The severe motor dysfunction with usually complete absence of sensory changes suggests that the lesion is in the spinal cord rather than in the peripheral nerve. Ferguson[3] reports congestion and round-cell infiltration in all parts of the brain of the child. Ross found these same changes most pronounced in the anterior and posterior horns of the spinal cord of the dog.[1] The absence of destruction of nerve cells is in keeping with the temporary nature of the paralysis. To determine whether the toxin acted peripherally, Ross exposed peripheral nerves in a paralysed and a normal dog, and found that the response to a faradic current was the same in each.[1] The site of action of the toxin therefore seems to be in the lower motor neurone, at least in cases of generalized paralysis.

The route by which the toxin reaches this site is not so plain, however. The likely routes are the blood stream and the axis cylinders of the peripheral nerves. If the spread is hæmatogenous, the toxin probably enters the lymphatics first and travels by them to the blood stream and so to the spinal cord. Slight enlargement of the lymph glands along the immediate drainage from the region of the bite is not unusual, though admittedly it may be due to slight superimposed bacterial infection in the bite. The fact that the legs are often affected first and most severely, even though remote from the region of the bite, suggests that the spread is hæmatogenous, the paralysis appearing first in the muscles that are being used most vigorously.

As for a spread along the peripheral nerves, it has long been accepted that toxins can proceed to the central nervous system by this route.[12] Marie produced suggestive evidence of this fact with tetanus so long ago as 1897. Subsequent work has shown that tetanus toxin is absorbed by motor

TABLE I.

Case Number.	Patient's Age.	Site of Attachment of Tick.	Length of Time Present before Paralysis.	Degree of Engorgement of Tick.	Site of Paralysis.	Duration of Paralysis after Removal of Tick.	Result.
I	2 months.	Right breast.	? 4 days.	1/2 to 2/3	General.	—	Death.
II	1½ years.	Scalp.	—	3/4 to full.	General.	4 to 5 weeks.	Recovery.
III	7 years.	Scalp.	? 4 days.	2/3 to 3/4	General.	5 weeks.	Recovery.
IV	7 years.	Right temple.	7 days.	—	Right side of forehead.	15 weeks.	Recovery.
V	10 years.	Scalp.	—	—	Legs.	2 weeks.	Recovery.
VI	5 years.	Scalp.	7 days.	Full.	General.	—	Death.
VII	10 years.	Right axilla.	10 days.	—	Right arm.	6 days.	Recovery.

nerve endings in the region of the wound, and passes along the axis cylinders of the peripheral nerves to the central nervous system. Occasionally, too, a purely local form of tetanus occurs. It has been proved also that neurotropic viruses can travel by the same route to the central nervous system. The close similarity between tetanus and tick paralysis in that each is caused by a toxin which spreads from a local wound to the lower motor neurones of the central nervous system to cause a generalized disturbance of motor function, but each of which may occur in a form localized to the region of the wound, inclines one to accept the view that the venom in each travels by the same route. It should not be unduly difficult to apply to tick paralysis the same experiments that have shown tetanus toxin to travel by the axis cylinders of the peripheral nerves.

Summary.

1. Tick paralysis, a disease known to occur in a number of domestic animals, is described as it occurs in humans.

2. The disease manifests itself as a widespread flaccid paralysis which may cause death by paralysis of the muscles of respiration.

3. Cases reported in the Australian medical literature are reviewed.

4. The tick responsible, *Ixodes holocyclus*, is described.

5. Treatment is discussed, including the treatment of patients with respiratory paralysis.

6. Seven cases which have occurred at the Royal Alexandra Hospital for Children are reported.

7. Reasons for the occasional localization of the paralysis are discussed.

8. The path by which the toxin reaches the central nervous system is discussed and the suggestion is made that it is the axis cylinders of the peripheral nerves.

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Reports of Cases.

"APOPLEXY" OF THE DEEP EPIGASTRIC ARTERY.

By J. M. DWYER,

Demonstrator in Pathology, University of Adelaide; Assistant Honorary Pathologist, Adelaide Hospital.

A WOMAN, aged eighty-six years, was attacked by a severe pain in the lower part of the abdomen at 2 o'clock in the morning. When seen six hours later she was still suffering from continuous pain, and she pointed to the lower left side of the abdomen as the site of its greatest intensity. She had felt sick, but had not vomited and had perspired freely. She was not short of breath, nor had she suffered from any of the symptoms or signs associated with infection.

On examination she was pale and shocked, but not unduly so. Her pulse was quite strong and regular, and not such as one would associate with the shock of a twisted ovarian cyst. Her tongue was clean and moist, and a general examination revealed a person in very good condition for her age. In the lower part of the left side a mass could be felt occupying a position such that its centre was about one and a half inches to the left of the middle line and about two inches below the umbilicus. Its appearance suggested that it crossed the middle line. On examination it was found to be fairly movable and appeared to be inside the abdomen. It was very tender, and, as far as the abdominal wall was concerned, did not appear to cross the mid-line. There was definite rigidity of the abdominal wall, and a sharp piercing pain on pressure suggested peritonitis, although the temperature of 99° F. did not suggest a peritoneal infection. A vaginal examination was not made.

At operation, after incision of the skin and subcutaneous fat, a bluish colour was discerned through the aponeurosis covering the *rectus abdominis*. On incision of the sheath of the rectus, blood clot became evident in the substance of the muscle, the fibres of which, though intact for the greater part, were separated into long strands, except at what appeared to be the focus of the bleeding, where the appearance suggested the destruction of a miniature explosion, the wreckage of muscle fibres being twisted and strewn about in all directions. At the focal point of this disturbance was found a ruptured deep epigastric artery. There was no evidence which suggested a rupture of the muscle substance by exertion or straining, and although this cannot be excluded absolutely, the appearances and history were against it, the ruptured artery being quite sufficient explanation of the appearances found. About sixteen ounces of blood clot were removed from the substance of the muscle, into which it had penetrated over an area about eight inches in diameter, with considerable bulging of the muscle sheath in all directions, particularly inwards towards the abdomen. This probably explains the peritoneal symptoms. The patient made an uneventful recovery.

Comment.

Mailer[1] reports a case and refers to rupture of the *rectus abdominis* as being the cause of this condition, usually in young untrained recruits, and states that spontaneous hæmatoma in women is far from common, Hilgenreiner[1] being able to collect only seven cases in 1924. I quote Mailer's description, which resembles the history of the case reported above.

> Interest arises from the fact, however, that it gives rise to symptoms and signs strongly suggestive of an acute intraabdominal condition, and is almost invariably diagnosed as such. There is a sudden onset with severe pain in the abdomen and evidence of peritoneal irritation in the form of vomiting, abdominal distension and some muscular rigidity. A swelling, tender on palpation, develops to the right or left of the median line, and most commonly below the umbilicus. Here the rectus muscle is narrower, is not reinforced by tendinous intersections, and below the semilunar fold of Douglas is devoid of posterior sheath, and rests directly on transversalis fascia. A hæmatoma in this situation therefore bulges towards the peritoneal cavity and is commonly mistaken for an ovarian cyst with some torsion of the pedicle, an acute appendicitis or an incarcerated para-umbilical hernia.

The case I have described suggests that the title I have given explains at least some of these cases.

Reference.

[1] Robert Mailer: "Spontaneous Hæmatoma of the Abdominal Wall", *The British Medical Journal*, Volume I, 1936, page 637.

Reviews.

AN INDEX OF TREATMENT.

THE twelfth edition of "An Index of Treatment", edited by Sir Robert Hutchison and Dr. Reginald Hilton, appeared last January.[1] This well-known volume has been published with the intention of providing practitioners "with a complete guide to treatment in moderate compass, and in a form convenient for reference".

Written by 78 contributors, all of whom are recognized authorities on their subjects, the work contains many excellent and interesting articles and is a mine of information in 1,000 pages. Its editors are to be congratulated on their compilation of this large volume.

However, like all omnibus collections, it suffers certain disadvantages. The editors could with advantage have made their contributors conform their articles to a more definite pattern and so could have avoided their obvious disparity in space and detail, some authors having written miniature monographs, whilst others have been contented with a mere précis. Devotion of much space to the detailed procedures of operations requiring special skill, such as harelip and bronchoscopy, seems to be a mistake, as these articles are too short for the man who will do this work, and too long to be merely informative. On the other hand, several minor complaints frequently met with are briefly dealt with, for example, boils and carbuncles. Surprise must be expressed that the disfiguring crucial incision for boils is still recommended and that faith in the local absorption of opium for piles still exists. Criticism in this directon may be taken much further, in that many cases might be quoted in which well-known, well-tried and useless treatments are still given, and even given pride of place, as first mention, where more modern discoveries have at least given something worth while. The impression is given in some cases that an advance in treatment has been tacked onto the end of an existing article, where rewriting of the old in the light of the new would have been better. Omission of certain well-recognized procedures also calls for comment—for example, the use of magnesium sulphate paste in carbuncles, sodium sulphate or colloidal kaolin in diarrhœa, and, above all, "M & B 693" in gonorrhœa. Disappointment must be expressed that the article on gonorrhœa was not rewritten in view of this epoch-making discovery, and the fact that it has received no mention as a treatment for this disease in a 1940 edition is unfortunate.

Despite the above criticism the volume contains an immense amount of valuable information and many excellent articles.

[1] "An Index of Treatment", by various writers, edited by R. Hutchison, Bt., M.D., LL.D., F.R.C.P., assisted by R. Hilton, M.A., M.D., F.R.C.P.; Twelfth Edition, revised; 1940. Bristol: John Wright and Sons Limited. Super royal 8vo, pp. 1011, with illustrations. Price: 42s. net.

THE SINGLE-HANDED MOTHER.

WE can commend "The Single Handed Mother", by Lindsey W. Batten.[1] The name of the book implies its nature; it "sets out to tell such mothers something of the natural history of babies and of the principles of mothercraft", and it is "intended to be read through rather than consulted stage by stage like a book of reference". It is seldom that a writer imparts so much practical information on the subject of mothercraft without fad or abstruseness; the book inspires confidence and problems rather fearsome become encompassable in the light and perspective of common sense. The literary style is good and the book reads well. The chapters deal with the baby in prospect, breast feeding, bottle feeding, weaning, management, bad habits, and problems and anxieties. The book would make an excellent yet inexpensive gift to a nurses' library or to the conscientious expectant mother who might need reassurance as well as information.

[1] "The Single Handed Mother", by L. W. Batten, M.B., M.R.C.P., with a preface by H. G. Wells; 1939. London: George Allen and Unwin Limited. Crown 8vo, pp. 188. Price: 5s. net.

PHARMACY, MATERIA MEDICA AND THERAPEUTICS.

NOTABLE changes have been effected in the thirteenth edition of Sir William Whitla's "Pharmacy, Materia Medica and Therapeutics", revised by Burn and Withell.[2] No attempt has been made to deal with the therapeutics of all pharmacopœial substances in alphabetical order, as in the 1933 edition, but the first third of the book is occupied by what might be described as topical therapeutics.

Modern views on diabetes are epitomized in an excellent article on carbohydrate and fat metabolism. Vitamins, which occupied a quarter page in 1933, run now to twenty-six pages. "Sex Hormones" gives recent knowledge of the action of œstrin, progestin and testicular extract. Iron, quinidine, ephedrine, "Coramine", "Cardiazol", the newer barbiturates in anæsthesia, chemotherapy by sulphanilamide and allied compounds, are all adequately described.

The book is improved by the omission of details of most drugs of doubtful value. The chapters on prescription writing and sterilization are most useful for both student and graduate. Unofficial remedies are omitted, and the book is smaller by one hundred pages.

This edition can be recommended, as could its predecessors, to all students and practitioners.

[2] "Whitla's Pharmacy, Materia Medica and Therapeutics"; Thirteenth Edition, revised by J. H. Burn, M.D., and E. R. Withell, B.Sc., B.Pharm., Ph.C., A.I.C., with a chapter on the use of vitamins in medicine, by H. M. Sinclair, D.M.; 1939. London: Baillière, Tindall and Cox. Crown 8vo, pp. 544. Price: 12s. 6d. net.

The Medical Journal of Australia

SATURDAY, JUNE 1, 1940.

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CHILDREN AND THE CINEMA.

ACCORDING to a recent calculation more than four million children in Great Britain go to the cinema at least once a week; the number who go oftener must run into many thousands. So far as can be discovered, the number of Australian children who visit the cinema is not known; it is probably safe, however, to conclude that it is relatively high. In any case the "pictures" have become so firmly established that they must be regarded as a normal form of entertainment for both young and old. That the scenes and actions depicted on the screen will have some sort of effect on those who see them few people will deny. Even those who look on any possible impression as purely momentary must admit that the cinema is informative and that individual members of an audience may be made aware of forms of behaviour previously unknown to them. Since many cinematograph films are obviously unsuitable for exhibition to children, there has arisen a problem that is the concern of all parents, of all teachers and of all who have any interest in the upbringing and general welfare of children. An important contribution to the solution of this problem has been made by Mr. Richard Ford in a book entitled: "Children in the Cinema", published in 1939 by George Allen and Unwin, Limited, of London.

It is not our intention to review this book in any great detail. Rather would we draw attention to several of its chief features, in the hope that those immediately concerned with the subject will study it as it deserves to be studied. One of the most important chapters is that dealing with psychological and physical aspects. The general conclusion is stated that for the vast majority of children the cinema is beneficial rather than harmful, provided care is taken in the selection of films. Those who understand the outlook of normal children will agree with the London County Council report that the morally questionable element in films is ignored by children of school age. Love-making on the screen bores the child—he does not understand it. But the drama that he does understand is real to him—he sits "in the presence of reality" and watches people who are in a real world, not in a world of make-believe. The child identifies himself with the character that he is watching. The author insists that this process of identification must not be mistaken for suggestion. The child is satisfied with the success of right over wrong and does not feel compelled to copy miscreants; some persons will not agree with this view. The overwhelming negative evidence presented by Mr. Ford in his discussion on the responsibility of films for child crimes support his contentions that the cinema must be absolved from blame. At the same time he is not over-insistent and states his case so that the reader may draw his own conclusions. The cinema, of course, is a "convenient scapegoat"; and we have known Australian magistrates to blame the cinema for delinquency without the slightest evidence to support their contention. Even if cinemas are partly to blame—and this has yet to be proved—it must be clear that they are never the whole cause of delinquency. The fundamental cause of delinquency lies, as one observer states, in the child's own attitude to his existence. Sir Samuel Hoare was undoubtedly right when he said that it was not so much films and shilling shockers that made juvenile crime, but broken homes, indulgent mothers, unkind stepmothers and unemployment.

Mention has been made of films that are unsuitable for children. To acknowledge that some films are not suitable for children is not incompatible with the view that they are not responsible for delinquency. This raises the question of censorship and the grading of films, both of which are in vogue. It also invites discussion on special film screenings for children. Mr. Ford has much stimulating information to give on this subject, and his book is worth reading from this point of view alone. He discusses teaching through entertainment, and makes an important point when he states that it is fantastic to suppose that children can be educated or improved in the cinema by methods which they can recognize as educational. In this they are like adults—or perhaps it would be truer to say that adults are like children in this respect. If cinematograph films are to be helpful to children—and this should surely be the objective of those who have to train the adults of the future—attention need not be confined to the film in relation to the child; the child may be considered in relation to the film. Mr. Ford has something to give his public on the teaching of film appreciation to children. This is a most worth-while adventure. Obviously if film appreciation is to be taught in schools, teachers themselves will have to become proficient in appreciation and in the art of teaching their pupils to think along the right lines. Film appreciation should probably be taught along with appreciation of music, of literature and of the art of living. This is not a Utopian dream, but a logical conception of what education should try to accomplish. Education consists in the "leading out" and the "building up" of the individual so that he can make the most of his privileges and opportunities—and no privilege or opportunity should be neglected.

Current Comment.

TUBERCULOSIS PRODUCED BY DEAD BACILLI.

ACCOUNTS have been appearing in French medical journals during the past year or two of researches in the bacteriology of tuberculosis made by A. Saenz and various collaborators in Uruguay. Recently Saenz has reported some noteworthy observations, which at first sight may seem scarcely compatible with our ideas of the pathogenesis of tuberculosis.[1] They are reminiscent of and comparable in interest with the now almost forgotten announcement of T. M. Prudden and Hodenpyl in 1891 that dead tubercle bacilli when injected into the body in sufficient numbers may produce nodules resembling tubercles.

With G. Canetti, Saenz injected into the testicles of rabbits some ten milligrammes of bovine tubercle bacilli, obtained from virulent cultures, killed by heat, washed and dried and suspended in one cubic centimetre of "oil of vaseline". The majority of the animals died within two months, whereas control animals injected by the testicular route with the oil alone or the bacilli in aqueous suspension or by the subcutaneous route with the bacilli in the oily suspension did not die. The testicles of the animals which died were found to be transformed into a great abscess, as might have been expected; but the lungs were affected extraordinarily. The lungs were considerably hypertrophied and scattered throughout them were great homogeneous patches of caseous necrosis, sometimes confluent, exactly as if the lungs were the seat of massive bilateral tuberculous pneumonia. The parts of the lungs not involved in the caseous process were hæmorrhagic. In other words, the disease produced was indistinguishable by inspection from massive bovine tuberculosis as it occurs in the rabbit from the inoculation of living bacilli.

It was naturally of great interest to study the effect of similar injections of human tubercle bacilli, which are scarcely at all pathogenic to the rabbit. The experiment was made and it was found that none of the animals was even made ill. When they were killed after several months it was found that their lungs were little, if at all, hypertrophied, that engorgement and rupture of blood vessels was little in evidence, while a few small whitish consolidated areas were peppered about, or else a zone of whitish consolidation was present over part of the surface of the lung, giving it a vaguely brain-like appearance. In short, the condition resembled that produced by the intravenous injection of a massive dose of living human tubercle bacilli. Further experiments were made with dead avian bacilli and with dead attenuated tubercle bacilli of various types, and analogous results were obtained. It is beside the present point to inquire how the dead bacilli in oily suspension reached the lungs from the testicles—that they did so was shown by the finding of acid-fast débris in the lungs—or to inquire what part the oil played in the pathological process set up in the lungs; the important fact is that dead bacilli in oily suspension injected into the testicles not only produced disease in the lungs of rabbits almost identical with that produced by living bacilli, but retained the degree and type of pathogenicity associated with their particular subspecies. Hence it is concluded that the pathogenic

[1] *Revue de la Tuberculose*, 1939-1940, Part 9.

properties of the various types of tubercle bacillus are bound up with their physical and chemical nature rather than with their metabolism and capacity to reproduce. It follows that the technique which has been devised is one which permits of study of the pathogenic properties of tubercle bacilli without consideration of the imponderable factors associated with their life and growth.

In the early days of bacteriology the power of a microbe to multiply in the body of a host was considered to be the measure of its virulence or disease-producing power. With the discovery of toxins it became necessary to speak of two main factors concerned in the production of disease: the multiplication of the living organisms in the host and the production by them of poisons affecting the surrounding tissues or the body generally. The capacity of dead tubercle bacilli to produce nodules resembling tubercles, noted by Prudden and Hodenpyl, was attributed to the presence of some slowly acting toxin contained in the bacterial bodies which became gradually diffused. The development of our knowledge of the phenomena of immunity and allergy, whereby the infected host is able by changes produced in itself to modify the course and virulence of an infection, and later the realization that different animals possess in different degree the capacity to manifest either or both of these phenomena, have greatly complicated studies in the virulence of tubercle bacilli. If the new laboratory method now presented to us makes it possible to nullify the factor of the multiplication of a microbe in our study of its pathogenic powers, then the revolution in our outlook on problems of virulence is complete.

APPLES IN THE PREVENTION OF LEAD AND ARSENIC POISONING.

"AN apple a day keeps the doctor away." We do not know where this important medical doctrine was first enunciated. But during the past twelve years over a hundred articles upon apples have appeared in the world's medical journals. Since A. Heiser revived the old raw apple cure for diarrhœa in Germany in 1928—it had been mentioned in an English book in 1775—and since E. Moro in 1929 described in detail the apple treatment of infantile gastroenteritis, the ripe and juicy apple has gained a particular niche in the orthodox *materia medica*. The literature on the subject was reviewed and the apple cure described in an article by P. A. Earnshaw in this journal on September 8, 1934. Since then many forms of dried apple have been placed on the market for medical purposes, and many theories have been put forward as to the mode of action of the apples. Some have thought that the therapeutic property resides in the malic acid, others have attributed it to albumin tannate; some have suggested that it may be due to the evolution of formic acid; others again have placed it in the pectin, and apple pectin has been used with more or less success in the treatment of diarrhœa.

Recently I. A. Manville, F. J. Reithel, P. M. Yamada, T. W. Spencer and J. R. Richardson[1] have made animal experiments that show a further remarkable property of apples, namely, that they appear to be able to prevent poisoning by lead arsenate. These authors have commenced a series of "studies in detoxication", and they begin by pointing out that certain noxious elements in the soil may get into plants in the same manner as essential elements, by solution, osmosis and diffusion; but the plant has chemical means of protecting itself against any possible harmful effect; for example, by the formation of glucosides. Similarly, in the animal body toxic elements gaining entry in food and drink may be inactivated or destroyed by some detoxicating mechanism, such as conjugation (although they may produce detrimental effects during their transit); but, in addition, some foods possess within themselves the ability to overcome many, if not all, of the toxic elements that they may naturally contain or may be made to possess artificially. Sulphur, calcium, glycine, pectin, sugar, vitamin *C* and the uronic acids have all been held to be important naturally occurring detoxicants. The authors have been especially interested in the metabolism of uronic acid, a product of the hydrolysis of hemicelluloses, and they have shown that the apple is a good source of this. They made their experiments with rabbits, guinea-pigs and rats, which were receiving a simple diet containing not much uronic acid. When the animals were thriving well, large doses of lead arsenate were added to the diet and some of the animals were given fresh ripe apple in addition. Animals that received the poison alone lost weight and many died; those receiving the apple as well as the poison were much less intoxicated and did not die. The rabbits, which received relatively more apple in proportion to the amount of lead ingested, were completely protected. The rats that received apple were much less intoxicated than rats that did not, despite the fact that their tissues, because their appetite was less impaired, contained more lead.

The authors promise the publication of further papers on detoxication, and state that bacterial as well as chemical poisons have been studied. The remainder of their important scientific studies and the news of their application to medicine will be awaited with interest. It is said that since the introduction of liver treatment for anæmia, liver has become almost unobtainable for the breakfast table, because the manufacturing chemists buy all the livers from the abattoirs; perhaps the day will soon come when all the apples grown will go to the manufacturers of uronic acid, and there will be no apple at playtime to keep the doctor away from Jack and Jill!

[1] *The Journal of Industrial Hygiene and Toxicology*, January, 1940.

Abstracts from Current Medical Literature.

DERMATOLOGY.

The Common Mole.

EUGENE F. TRAUB AND HARRY VEIL (*Archives of Dermatology and Syphilology*, February, 1940) discuss a basis of classification for moles and attempt to establish some clinico-pathological criteria on which some estimate of the likelihood of malignant degeneration can be formed. After discussing the definition of the term "nævus" and the varying lesions described as "moles", the authors define as a mole a cellular nævus characterized histologically by the intracutaneous arrangement of nævus cells in special configurations (nests, bands and strands). Nearly all these lesions contain a variable amount of melanin and may be present at birth or appear in later life. In the authors' opinion, unlike that of Unna, there is no evidence that these nævus cells arise from the epidermis. The term melanoma is restricted by the authors to a lesion thought to be potentially or actually malignant. The type of lesion which the authors consider likely to become carcinomatous is that in which the origin of the lesion can be demonstrated histologically at the epidermo-dermal junction. These lesions are smooth, devoid of hair, and vary in colour from brown and blue to black. They are usually found on the extremities, especially the lower, though the face may be affected. This is the type of lesion in which tampering or incomplete removal is dangerous. The term nævo-carcinoma is restricted by the authors to lesions in which previously existing nævus cells become cancerous. In the opinion of the authors this does not occur; but they consider that the malignant change takes place at the epidermo-dermal junction. In other words, the lesion is a melanoma superimposed on a pre-existing common intradermal mole—a rare condition in the authors' opinion. In this regard they have frequently looked for lesions on the lower extremities which could be regarded as common moles, but could find none. Two important points are stressed. In a pigmented lesion of the lower extremity the structure found was that of a growth originating from the epidermo-dermal junction (referred to by the authors as junction type nævus or potentially malignant melanoma). Secondly, lesions of the face, though frequently irritated, cut or incompletely removed, never, in the opinion of the authors, have given rise to malignant growths. Regarding lentigines, the authors consider the superficial variety to be potentially malignant. The junction type nævus of the authors corresponds to the process of *Abtrofung* described by Unna, who, however, believed, unlike the authors, that this "dropping" of cells at the junction zone was the origin of true nævus cells. The authors discuss also the difficulty in diagnosis which may arise in numerous small lesions of the skin which may resemble the common mole. In evaluating the results of a *questionnaire* submitted to about 120 members of the American Dermatological Association, the authors state that out of 93 persons who replied, 85 had not observed malignant change in a common hairy mole nor had a histologically proved benign cellular nævus subsequently become malignant. The authors conclude by setting out in tabular form a classification of moles into infraepidermal, intradermal, junction or borderline combination type and blue nævi. The last-mentioned is a mesodermal growth containing pigment and, although often mistaken for a melanoma, is, in the authors' opinion, innocuous. The paper concludes with a detailed series of case records, including clinical and histological photographs.

The Influence of Age on Tinea Capitis.

LYLE B. KINGERY, in collaboration with Roger J. Williams and H. A. Kidd (*Archives of Dermatology and Syphilology*, December, 1939), describe an experimental study made to investigate the well-known phenomenon of the rarity of scalp ringworm in persons past the age of puberty. Briefly, their work is a very well-planned laboratory experiment which demonstrates the different amounts of fungus growth on a medium inoculated with an aqueous extract of pooled hair obtained from subjects of varying age groups. The culture medium was a special one to eliminate the error due to the effect on culture growth obtained according to the quantity of fungus inoculated, this error being a common one in bacteriological work generally. The medium used enabled a constant rate of growth to be obtained with a minimal concentrate of fungus spores, this concentrate itself being checked by the hæmocytometer. The hair extract was obtained from samples of hair of three age groups: five, ten and over twenty-five years. Each group of samples was thoroughly mixed and cleaned before a sterile aqueous extract was made. Fungus cultures were made on the medium inoculated with hair extract from each group. A non-inoculated control was employed. Results were estimated at first by dried weight of fungus growth, but later estimation by eye proved quite satisfactory. Several types of fungus were used, namely, *Microsporum audouini*, *Microsporum lanosum* and *Microsporum fulvum*. Several differences were seen owing to the individual nutritional requirements of each fungus. The results of the experiment, though showing some inconsistencies, demonstrated a more rapid fungus growth on the medium containing "young" hair extract than on those to which extract from "old" hair had been added, or on the control medium.

Impetigo Contagiosa.

MORTON SELDOWITZ (*American Journal of Diseases of Children*, January, 1940) describes a new method of treating *impetigo contagiosa*, and discusses the results of the treatment of a series of 38 children suffering from this skin infection. Briefly, the treatment consists of the application of 8-hydroxyquinoline in a rubber base (containing 3% boric acid and 6% magnesium carbonate). The patients were divided into two groups, one being composed of very young children suffering from the bulbous form of the disease, the other being a series of older children with the more common confluent crusted variety. The technique consisted of the application of the compound after a thin layer of gauze had been placed over the lesions, care being taken not to disturb mechanically the overlying crusts. The dressing was fastened in place with adhesive plaster and was changed at intervals of two to four days. The gauze was used to absorb any exudate caused by liquefaction of the crust or from the lesion itself. In each group a number of patients were treated direct by the new method, whilst others, as controls, were previously treated by the more common methods, by ammoniated mercury ointment, gentian violet, dry gauze or dry heat. Other controls were dealt with by application of the rubber base without the addition of the 8-hydroxyquinoline. The value of the treatment consisted not only in rapidity of healing, but in the lack of spread which frequently occurs under other occlusive methods. One patient only showed spread; but this was due to detachment of the dressing and scratching. In three out of the 38 patients the treatment gave unsatisfactory results. One patient responded to each treatment, but relapsed after some weeks' interval. This occurred several times and was considered to be due to a general lowering of resistance to infection. In the other two patients the lesions were examples of impetigo supervening on an extensive and severe dermatitis involving large areas of the trunk and extremities. The author suggests that it would appear that secondary impetiginization of an allergic cutaneous condition, such as eczema or papular urticaria, does not respond so well as primary impetigo.

UROLOGY.

Early Cases of Renal Tuberculosis.

H. WILDBOLZ (*Report of VII Congress of the International Society of Urology*, 1939) holds that until recently practically the only early cases of renal tuberculosis detected

clinically were those in which the lesions, though very small, were visible to the naked eye. Today, microscopic examination has led to the detection of still earlier lesions. It has now been shown that tuberculous bacilluria indicates an actual tuberculous lesion of the same kind in the kidney, if the bacilli are collected by a ureteric catheter. The tuberculous nature of the bacilli must be proved by culture and animal inoculation. The most common form of early renal lesion is the caseous type, in which the lesions are in the medullary portion, particularly in the papillæ or calyces. Tendency to caseation and to involvement of the renal pelvis is early. The second or less common type is the fibrous variety, in which the early tuberculous lesions are mostly in the renal cortex and show an early tendency to fibrous atrophy rather than to caseation. There is a third early form, of rare occurrence, in which tubercle bacilli enter the kidney tissue and become attached, without producing tubercles. An allergic reaction of the tissues brings about rapid destruction of the bacilli, and as a result no tubercles are formed, but only a non-tuberculous inflammation. A bacilluria demonstrable through animal inoculation and culture seems to occur rather early after the onset of a tuberculous renal infection. Therefore, bacteriological examination of the urine makes diagnosis possible in the earliest stage. On the other hand, it may be impossible for a rather long time to determine the type of renal tuberculosis present. It is important to distinguish the caseous from the fibrous type, since the former is rarely curable, while the latter is curable with retention of organic function. Typical renal pyuria and rapidly decreasing secretion of the disabled kidney indicate the probable presence of caseous renal tuberculosis. On the other hand, if the urine from the bacilluric kidney remains free from pus for a long period, or exhibits only a few leucocytes and its efficiency is only slightly impaired, the fibrous form of renal tuberculosis is most probable. Immediate nephrectomy is indicated if a caseous renal tuberculosis seems probable, but when the fibrous type is probably present nephrectomy should be discouraged. A general non-surgical tuberculosis treatment should be adopted, but during this period an accurate urinary control of the patient for four to six months is necessary. In early bilateral renal tuberculosis nephrectomy is indicated only when in the course of years the stage of the disease in the two kidneys differs to a great degree.

Urethro-Deferential Reflux.

J. PERVES AND H. DUVERGEY (*Journal d'Urologie*, August, 1939) discuss the phenomenon of reflux of fluid (urine, semen or any injected substance) from the male urethra into the seminal vesicle through the canal of the *ductus deferens*. Normally the orifices of the ejaculatory ducts are physiologically closed and are impermeable to any fluid injected into the urethra. This can be proved by studying urethrograms made after the injection of radio-opaque substances into the urethra. No muscular sphincter protects these orifices, but they are narrow and obliquely placed. Under certain dynamic conditions, however, this reflux occurs and almost certainly explains the occurrence of epididymitis. Certain classic cases are quoted which definitely prove the existence of a reflux of urine, particularly one in which fistulæ emitting urine followed on the performance of bilateral vas section. The emission of urine from the *ductus deferens* occurred at each act of urination. The authors illustrate their description of this phenomenon by reporting four personal cases. Two of them are concerned with the occurrence of epididymitis, one of which was definitely tuberculous; the remaining two cases occurred after prostatectomy. In the former two cases, ureterography with lipiodol demonstrated this reflux, the lipiodol flowing back along the *ductus deferentes* and demonstrating lesions in one or both seminal vesicles. In the post-prostatectomy cases, reflux was observed passively in one case, that is, during ordinary urethrography, while in the other case the reflux occurred actively, that is, during micturitional urethrography. From these observations it can be taken as proved that, in certain circumstances, the deferential lumen is permeable to liquids. It may therefore be assumed that it is just as possible for germs to follow the same route.

Theories of Calculus Formation.

J. SWIFT JOLY (*Urologic and Cutaneous Review*, October, 1939) reviews the current theories of stone formation, pointing out that a new theory is favoured by each succeeding decade. (i) Diathesis is considered to be responsible for the formation of cystin calculi, but not those of other chemical constitution. It is erroneous to believe that the presence of a crystalline deposit of a stone-forming salt in the urine is due to diathesis, nor does it prove that such salt is excreted in excess. It is due to the fact that the urine is temporarily unable to hold that amount of salt in solution. (ii) Hyperexcretion was considered a likely cause when it was discovered that stones were composed of substances ingested (phosphates and oxalates) or resulted from metabolism of protein (urates, cystin). Some stones are so formed, for example, urate stones in those who overeat, cystin stones in persons suffering from cystinuria, and phosphatic concretions in patients with hyperparathyreoidism. A world-wide view of calculus disease, however, shows that it is rare in districts in which the diet is varied or where large quantities of dairy products are consumed, whereas it is prevalent among those who subsist on a poor, monotonous or ill-balanced diet. For example, natives of Asia's stone-forming districts live on a purin-free diet without excess of oxalates, yet the stones formed are chiefly urates and calcium oxalate. (iii) In regard to the protective action of colloids, the author believes that the colloids play a large part in the maintenance of the condition of supersaturation of the urine by certain salts. Failure of this mechanism could be brought about by the fusion of minute colloid particles—which is known to occur commonly in colloid suspensions—thus diminishing surface area and surface energy. Thus the sparingly soluble particles crystallize out on the colloid particles and achieve their characteristic forms. (iv) Vitamin deficiency is held to be one of the major factors in stone formation. The prevalence of vitamin deficiency in England, Europe and America has escaped attention until recently. The author considers this an important factor, but not the only one. (v) In regard to mineral balance, earlier teaching advised the reduction of the intake of calcium to avoid stone formation. It has now been shown that the urinary excretion of calcium varies inversely with the intake, particularly if there is, in addition, a deficiency of vitamin *D*. Elimination of calcium is also influenced by the amount of magnesium present in the urine, the variation being in inverse proportion. The author quotes the work of Hammarsten, in which she not only produced stones in laboratory animals, but subsequently decalcified them by dietetic means. He concludes that with a diet rich in vitamins, calcium and magnesium the risk of stone formation is at its lowest.

Large Vesical Diverticula.

R. W. BARNES (*Journal of Urology*, November, 1939) presents a simplified technique for the surgical ablation of large vesical diverticula situated deeply in the pelvis. The bladder is widely opened suprapubically and the diverticular orifice is exposed. The ventral portion of the diverticular sac is then exposed extravesically. An incision is made in the bladder wall from the cystotomy opening to the orifice of the diverticulum and continued through the ventral wall of the diverticulum. The mucosa is then incised at the orifice, whence it is stripped from the diverticular cavity. In some places, particularly in the depths of the cavity, the mucosa is so densely adherent that it tears. With such good exposure the edge may be readily identified and picked up with forceps. Where it is impossible to strip it off, a cautery is used to destroy the remainder. The diverticular orifice and incised bladder wall are then closed in two layers with drainage. The author has been gratified by the absence of shock and infection in complicated cases, as compared with the operation of radical diverticulectomy in similar cases.

British Medical Association News.

SCIENTIFIC.

A MEETING of the Victorian Branch of the British Medical Association was held at the Women's Hospital, Carlton, Melbourne, on April 17, 1940. The meeting took the form of a series of clinical demonstrations by members of the honorary medical staff of the hospital.

Chorionepithelioma of the Fallopian Tube in Extrauterine Pregnancy.

DR. EDWARD WHITE showed a patient suffering from primary chorionepithelioma of the Fallopian tube in an extrauterine pregnancy. She was aged twenty-six years and had been admitted to hospital three weeks earlier with acute abdominal pain and vomiting. She had had one child seven years earlier, and two years prior to the meeting left salpingectomy had been performed for a previous ectopic pregnancy.

On examination she was pale and her pulse rate was 100 per minute. Tenderness was present about the cervix and right fornix, but there was no palpable mass. The diagnosis was a ruptured extrauterine pregnancy on the right side. An immediate operation was performed, and this disclosed a gestation sac in the right Fallopian tube, which had ruptured, causing a considerable amount of hæmorrhage into the abdominal and pelvic cavities. Right salpingectomy was performed, but the ovary was retained. After gentle clearing of the abdomen and pelvis of blood and clot the patient was given one pint of citrated blood by transfusion. She made an uneventful recovery.

A histological examination disclosed clear evidence of chorionepithelioma in the pregnant tube. Dr. White remarked that that was a very rare condition. He said that, on account of the youthfulness of the patient, deep X-ray therapy would be withheld as long as regular and frequent Aschheim-Zondek tests continued to yield negative results.

Retroperitoneal Hæmorrhage Simulating Ruptured Extrauterine Pregnancy.

Dr. White's second patient was suffering from retroperitoneal hæmorrhage, simulating ruptured extrauterine pregnancy. She was aged twenty-two years, and had been admitted to hospital in a state of severe shock and collapse. There was a history of severe abdominal pain on the left side of six hours' duration, and of vomiting and amenorrhœa of eight weeks' duration. Tenderness was present about the cervix. A provisional diagnosis of severe internal hæmorrhage from a ruptured ectopic pregnancy was made.

At operation an extensive retroperitoneal hæmatoma was found on the left side, behind the descending colon and the base of the mesentery. There was an intrauterine pregnancy of about eight weeks. The abdomen was rapidly closed whilst blood transfusion was proceeding. Later it was found that the patient had fallen down some steps two weeks earlier and had severely injured her back and left side. The retroperitoneal hæmorrhage was probably traumatic in origin. She made an uninterrupted recovery.

Pregnancy Complicated by Impacted Myoma in the Pelvis.

Dr. White's third patient was aged thirty-one years. She had had severe colicky pain for thirty-six hours prior to her admission to hospital.

On examination she was found to be five months pregnant, and a large myoma, the size of a cricket ball, was firmly fixed in the pelvis under the sacral promontory.

At operation an interstitial myoma was lifted out of the pelvis and then dissected out of the uterine wall. Examination of the myoma disclosed early red cell degeneration. The patient was given narcotics freely, and received daily injections of *corpus luteum* extract. Her convalescence was normal and the pregnancy was uninterrupted.

Abortion and Fatal Spirochætal Jaundice.

DR. J. W. JOHNSTONE discussed the history of a patient, a *multipara*, aged thirty-four years. She was ten weeks pregnant when admitted to hospital, and confessed to having taken medicines and used a douche one week previously to procure an abortion. When seen she was very ill; an abortion had occurred twelve hours previously. The outstanding clinical features, apart from the severe toxic state, were high fever, muscular pain, deep jaundice and petechial hæmorrhages, which on the face became confluent into a large ecchymotic mask. Death occurred on the seventh day with asthenia and renal failure. Ordinary bacteriological investigations on material from the genital tract, throat, urine and blood failed to reveal *Bacillus welchii*, hæmolytic streptococci or other organisms of significance, nor was there any evidence of metallic poisoning. Dr. Johnstone explained that the case was first thought to be one of *Bacillus welchii* infection, the clinical features and particularly the facies of the patient being practically identical with those found in many of the gas infections seen at the Women's Hospital. One point of difference was the absence, in the case under discussion, of hæmolysed blood pigments in the urine. When investigations had failed to reveal the presence of *Bacillus welchii* or other significant organisms, the case became an interesting diagnostic problem.

The *Leptospira* organism was demonstrated by dark-field examination in the patient's blood and urine during life, and also in the tissues *post mortem*. Bacteriological investigations had not yet been sufficiently completed to make it certain that the *Leptospira* in this case was the typical *Leptospira ictero-hæmorrhagiæ* or a related variant. However, the clinical manifestations illustrated many of the features of the more severe form of Weil's disease, which was recorded as being more common during pregnancy; the mortality rate was then high and abortion was a likely occurrence. Dr. Johnstone thought that, in spite of the patient's history of having taken drugs and of interference by douching one week previously, the specific organism had found entry by means other than the genital tract and the disease had caused the abortion.

Spirochætes of the ictero-hæmorrhagica group appeared to be endemic in rats and to be disseminated by their urine. In areas where man and rats become intimately associated, such as the Queensland canefields, in Japan, and amongst sewer workers, the disease was frequent. Abortion from a sporadic infection, such as had occurred in the case under consideration, must be a rare event in Melbourne. Although the patient came from the recently built "Garden City" area of Port Melbourne, it was pointed out that that district was in close proximity to the dock area.

Dr. Johnstone illustrated his description by a demonstration under dark-ground illumination of the *Leptospira ictero-hæmorrhagiæ* taken from an infected rat caught in the fish market, and also by a projected colour photograph of the patient's face. This showed clearly the combination of deep jaundice and gross ecchymosis, which made the patient's facies one of the outstanding features of the case.

Congestive Heart Failure.

PROFESSOR MARSHALL ALLAN showed a patient, aged twenty-eight years, who had been in hospital eighteen months previously with cardiac failure; this had occurred towards the end of her first pregnancy. There was no history of rheumatic fever or of any other acute infection. After compensation had been attained she was delivered by Cæsarean section, and sterilization was performed by clamping and ligature of the tubes with catgut.

She had presented herself again two weeks prior to the meeting with a history of severe dyspnœa and orthopnœa, palpitation and œdema of the ankles. A severe degree of mitral stenosis was present and there were symptoms of an adherent pericardium. No adventitious sounds were heard in the lungs. The liver was palpable

below the costal margin. The *fundus uteri* was at the level of a sixteen weeks' pregnancy. After rest and medicinal treatment abdominal hysterotomy was performed and the tubes were resected. The first three days of convalescence were stormy; but the patient's general condition was now fairly good, though considerable dyspnœa was still present. Professor Allan said that this case illustrated the need to terminate a pregnancy occurring in a patient with a history of recurrent heart failure. Although the Madiener technique for sterilization was said to yield good results, it was not infallible, and this was the second failure which Professor Allan had seen during the past year.

Pulmonary Tuberculosis.

Professor Allan also showed two patients suffering from tuberculosis associated with pregnancy. The first was a *primigravida*, aged twenty-one years, with a history of infection of the upper lobe of the left lung, noted two years earlier. Artificial pneumothorax had been performed and the thoracic cavity had been refilled with air at regular monthly intervals since. The patient was first seen in the antenatal clinic six weeks prior to the meeting, when she was fourteen weeks pregnant. The left side of the chest was hyperresonant, and breath sounds were diminished at the apex of the left lung and absent at the base. No adventitious sounds were heard. The right side was normal. An X-ray examination revealed a partial pneumothorax on the left side with basal adhesions. The patient was seen by a consultant and arrangements were made for a further course of pneumothorax therapy.

The second patient was a *multipara*, aged twenty-five years, whose last confinement had occurred eighteen months earlier. She gave a history of frequent coughs and colds since July, 1939, and of having lost eight pounds in weight during the past month. A severe attack of hæmoptysis caused her to seek advice when she was five months pregnant.

On her admission to hospital her temperature was 99·4° F. and her pulse rate 124 per minute. Dulness to percussion was elicited in both apices, especially the right, with prolonged expiration and a few crepitations and rhonchi. No abnormality was detected in the left side. Examination of the sputum revealed many acid-fast bacilli, and an X-ray examination of the chest revealed a recent tuberculous infiltration of the upper half of the right upper lobe. Rest in bed and sedatives rendered the cough less severe, and the sputum was free from blood after a few days. The patient's general condition had also improved. Arrangements had been made for her admission to a sanatorium for artificial pneumothorax.

Professor Allan discussed the modern trends in the treatment of pulmonary tuberculosis during pregnancy, and pointed out that conservative measures had now been adopted largely instead of interruption of the pregnancy. Such treatment was advocated even in the early months of pregnancy; but to be of any value the patient must be under constant sanatorium supervision, and this was not always easily arranged.

Toxic Nodular Goitre.

Professor Allan then showed a patient, aged thirty-six years, who was a *multipara* with three children; the youngest was three years old. No enlargement of the thyreoid gland had been noted during these pregnancies. About a year prior to the meeting she complained of a swelling in the neck associated with general debility, and attended a general hospital for five months. She was then found to have become pregnant, and was referred to the Women's Hospital for advice regarding the pregnancy. The thyreoid gland contained a large nodule in the left lobe. She was considered a suitable subject for operation, which had been performed at the general hospital four months earlier. At the time of the meeting she was near term and had improved in general health, although anæmia was still a pronounced feature, despite the use of iron tonics.

Professor Allan discussed the relationship of the various forms of goitre and pregnancy. He emphasized the need not to treat patients suffering from toxic goitre with iodine for too long a period. If no improvement had been noted within a short time, thyreoidectomy should be performed. The pregnancy should not be interfered with unless the patient was near term, when premature induction of labour was preferable, and the operation could be postponed until after the puerperium.

Korsakoff's Syndrome following Hyperemesis Gravidarum.

The next patient shown by Professor Allan was aged twenty-two years; she was a *multipara*, whose last pregnancy had occurred a year ago. Her medical attendant had sent her to hospital with a letter stating that she had suffered from severe hyperemesis for several weeks and was now mentally confused.

On admission to hospital she appeared fairly well nourished, and all vomiting had ceased. The urine was clear, but gave an acetone reaction. She appeared to be very apathetic and confused, could not remember the day or the month, and insisted that she had been delivered. The knee jerks were equal and exaggerated; there was no sign of jaundice and the position of the fundus corresponded to an eighteen weeks' pregnancy. Sedatives and injections of a vitamin B_1 preparation ("Vibex") were given, and saline solution was administered intravenously. The patient continued to be noisy and irrational, and after a few days nystagmus was noted. Examination of the ocular fundi failed to reveal anything of note. Lumbar puncture revealed no increase of pressure and the chloride content of the cerebro-spinal fluid was low, while various tests revealed no abnormality. The next development was numbness of the fingers followed by slight foot-drop and wrist-drop. In addition to the vitamin B_1 injections other vitamin preparations, such as "Marmite" and substances containing vitamin *A*, were given. At the time of the meeting, while the patient's general condition seemed to have improved, the mental state was unchanged. Termination of the pregnancy was refused on religious grounds.

Professor Allan referred to the need for administration of vitamin B_1 preparations in particular in all cases of vomiting in which intravenous therapy had been used for more than four or five days. The prognosis in such cases must be guarded. He had encountered two similar cases in which both mental and neuro-muscular symptoms had still been present more than a year after the termination of the pregnancy.

Blood Pressure in Pregnancy.

DR. GEORGE SIMPSON showed three patients illustrating the three types of blood pressure found in pregnancy. They illustrated: (i) essential hypertension in pregnancy, (ii) nephritic toxæmia, and (iii) the "early warning rise" of blood pressure that in the early months of pregnancy indicated the possibility of preeclamptic toxæmia or of eclampsia later. Dr. Simpson stressed the importance of taking blood pressure readings at antenatal visits, and demonstrated a mercurial sphygmomanometer on a stand, with which this could be done quickly and accurately.

Bacteriological Demonstration.

MISS HILDRED BUTLER demonstrated tests for the determination of virulence of strains of *Streptococcus hæmolyticus* group A. These included the staining of capsules and the type of growth in serum broth and on neopeptone-blood-agar plates. The practical importance of the coagulase test in differentiating between pathogenic and non-pathogenic staphylococci was also demonstrated. Miss Butler also showed smears and guinea-pig inoculations illustrating the rapid identification of *Vibrio septique*. Two strains recently isolated from fatal cases of septic abortion produced the characteristic picture in the guinea-pig within eight hours of inoculation.

Significance of the Aschheim-Zondek Test in the Early Diagnosis of Chorionepithelioma.

DR. H. F. BETTINGER, in conjunction with DR. W. G. CUSCADEN, showed a patient to illustrate the significance

of the Aschheim-Zondek test in the early diagnosis of hydatidiform mole and chorionepithelioma. This case will be reported in detail in another issue.

Pathological Demonstration.

Dr. Bettinger then discussed the case of a woman, aged twenty-four years, who, after a normal pregnancy, suddenly had an eclamptic fit. She was admitted to hospital shortly afterwards and was found unconscious; severe œdema of the lower extremities was present, and the systolic blood pressure was 126 millimetres of mercury. Lumbar puncture was performed and almost pure blood was obtained. The patient died seven hours after her admission to hospital, undelivered and without regaining consciousness. The post-mortem examination revealed a typical eclamptic liver and a subarachnoidal hæmorrhage surrounding the pons and the *medulla oblongata*. The compression of the vital centres by this hæmorrhage accounted for the rapid course of the disease and the death of the patient.

Dr. Bettinger then discussed the case of a woman, aged forty years. She was reported to have had congenital heart disease, the exact nature of which had never been determined. Subtotal hysterectomy and bilateral salpingo-oophorectomy were performed for fibromyomata of the uterus and endometrioma of the ovary. During the week following the operation her condition became gradually worse, and death followed the signs of heart failure. At the post-mortem examination it was found that the main stem and the two main branches of the pulmonary artery were considerably dilated; they showed pronounced atheroma and calcification. The right ventricle was hypertrophic. The *ductus arteriosus* was patent, and atheroma and calcification were noted in it. Dr. Bettinger pointed out that the condition of the pulmonary artery was certainly due to the patency of the *ductus arteriosus*. In normal circumstances the pressure in the pulmonary artery was considerably lower than that in the aorta; but with a patent *ductus arteriosus* the pressure in the pulmonary artery had to be as high as that in the aorta, because otherwise blood would flow continuously from the aorta through the duct into the pulmonary artery. This rise in the blood pressure in the pulmonary artery accounted for the changes found in the pulmonary artery and in the right ventricle. The post-mortem examination revealed some other very unusual features. Extending from a thrombosis of the left ovarian vein there was a thrombosis of the left main renal vein and the majority of their branches, which resulted in a hæmorrhagic infarction of the whole of the kidney. From this thrombosis a paradox embolism had occurred through the patent duct into the spleen.

Dr. Bettinger next discussed a dysgerminoma of the ovary. He said that the tumour had been found in a woman, aged twenty-six years. It was obstructing labour. During pregnancy a provisional diagnosis of twins had been made. The X-ray examination did not confirm that diagnosis, but suggested somewhat the presence of a teratomatous growth. The tumour removed had the typical structure of a dysgerminoma (seminoma). Dr. Bettinger said that the occurrence of such a tumour during pregnancy was rather unusual, as these tumours were mostly found in persons with malformation or under-developed genital organs.

Dr. Bettinger then showed specimens and described a case of hydranencephaly. This case will be reported in detail in another issue of the journal.

The next two cases discussed by Dr. Bettinger were cases of diaphragmatic hernia. He said that the majority of such cases were not true cases of hernia, because usually there was merely a defect of the diaphragm, through which abdominal organs entered the thoracic cavity, but not a peritoneal sac, as in all other forms of hernia. A specimen of false hernia and one of true diaphragmatic hernia were shown.

Dr. Bettinger went on to describe several cases of congenital heart disease. The first two patients showed a transposition of the aorta and the pulmonary artery; both these children had lived for about four weeks. Life was possible under such a condition only by the passage of blood from the left ventricle through the pulmonary artery and a patent *ductus arteriosus* into the aorta. When such children had one malformation more—namely, a defect of the ventricular septum—life was possible even for a number of years. In Dr. Bettinger's next two cases a very uncommon condition was present. In both the aortic os of the left ventricle was completely obliterated. The pulmonary artery was more than twice the normal width; the ascending aorta and the arch of the aorta were extremely narrow; the *ductus arteriosus* was patent. Dr. Bettinger pointed out the alterations of the circulation which had to take place under such a condition. In one case endocarditis of the mitral valves was found, and this might indicate that the condition was due to intrauterine inflammatory processes. The last case was one of dissecting aneurysm of the *ductus arteriosus*, which had perforated into the left bronchus. This case will be reported in detail in a later issue of the journal.

Abdominal Tumours.

DR. W. I. HAYES showed three patients suffering from abdominal tumours, two of which were large mid-line tumours due to a fibroid condition of the uterus; the other was a tender mass in the right iliac fossa, believed to be a carcinoma in the transverse colon.

A woman, aged forty years, who had borne two children, had had menorrhagia for a year and complained of shortness of breath and weakness, especially after the menstrual periods. The excessive loss of blood with clots had caused serious anæmia, the hæmoglobin value having been reduced to 50%. She had noticed for six months that the abdomen was growing larger.

The other patient suffering from a fibroid condition of the uterus was aged forty-three years, and she had had one successful pregnancy seventeen years earlier. She had had no dysmenorrhœa, though the menstrual discharge was profuse and dragging intermenstrual pain of an intermittent nature had been present in the right iliac fossa. For four months she had been feeling weak and the tumour had grown until it almost reached the level of the umbilicus.

The third patient was aged seventy-two years, the mother of eleven children. She had reached the menopause when aged fifty years. For three months she had had continuous dull aching pain, localized in the right iliac fossa. Her appetite had failed during the preceding six weeks, with resultant loss of weight, and the stools for the past two months were described as "slimy". There had been no vaginal bleeding or discharge. The systolic blood pressure was 126 and the diastolic pressure 85 millimetres of mercury. On examination a mass could be felt in the right iliac fossa, and on the skiagrams it appeared probable that the condition was a malignant tumour affecting the transverse colon.

Post-Abortional Pelvic Peritonitis.

DR. J. A. H. SHERWIN showed two patients suffering from post-abortional pelvic peritonitis, to illustrate the effect of early and delayed treatment of this condition with short-wave diathermy. One of the patients had been acutely ill, with a high temperature and rapid pulse rate; short-wave therapy was commenced three days after her admission to hospital. Very shortly the symptoms abated; ten days later her pulse rate and temperature were normal and the patient was making good progress towards recovery. The other patient had also been acutely ill. The treatment was not commenced till fourteen days after her admission to hospital. Subsidence of the fever and general improvement were much more rapid than in the previous case; her pulse rate and temperature fell to normal in forty-eight hours. Dr. Sherwin said that the object of the demonstration was to show that the most severe types of acute pelvic inflammation with accompanying peritonitis might be treated by short-wave therapy from the outset with good effect.

Fibroid Tumours.

DR. A. M. WILSON and DR. W. M. LEMMON showed two young women suffering from fibroid tumours. The chief symptom in each case was menorrhagia, and both patients had had an intensive course of endocrine therapy. Examination under an anæsthetic revealed an enlarged uterus, and laparotomy enabled a submucous fibroid tumour to be removed in each case.

Prolapse of the Vaginal Vault following Hysterectomy.

DR. N. LENNOX SPEIRS showed a patient suffering from prolapse of the vaginal vault following vaginal hysterectomy. He said that this unfortunate complication occurred fairly frequently, and he discussed possible means of minimizing the chances of its occurrence.

Prolapse of the Uterus.

DR. GEORGE BEARHAM showed two young married women with second-degree prolapse of the uterus, and discussed the possible methods of treatment. He said that both had some stress incontinence of urine, which seemed to indicate that surgical measures were necessary.

A Respirator for the Treatment of Asphyxia Neonatorum.

PROFESSOR AUBREY BURSTALL, of the School of Engineering, University of Melbourne, demonstrated a respirator of his own design for the treatment of asphyxia in the newborn. The machine was constructed in the engineering workshops at the University of Melbourne, and was then undergoing trial at the Women's Hospital. The apparatus was similar in principle to that made for the same purpose by the Drinker Company of the United States of America, but it differed in that the child was housed in a hot-water jacketed cylindrical chromium container. The apparatus was normally operated by electricity, but could also be worked by hand. It had been used in the treatment of several cases at the hospital with very promising results.

The Management of Disproportion.

DR. W. D. SALTAU showed three patients to illustrate the management of disproportion. The first was a *primipara*, aged twenty-one years, whose expected date of confinement was March 8, 1940. Her stature was very small; she was only four feet seven inches in height. Her external measurements were: interspinous, nine and a half inches; intercristal, ten inches; and external conjugate, seven and a quarter inches. The sacral promontory was palpable. There was no noteworthy abnormality during the antenatal period, except that the fœtal head remained high and showed no signs of becoming fixed. She was admitted to hospital on February 29, a few days before the due date, for medicinal stimulative treatment. On the first occasion this was unsuccessful; but two days later labour commenced in response to a second course. She had a few contractions about midday on March 2. These were of moderate strength until about 8.30 p.m., when the membranes ruptured. The head was still unfixed. She had strong contractions until midnight, when the head had become fixed, but the greater part was still above the pelvic brim. A vaginal examination was made at 12.30 a.m.; the cervix was found to be three-quarters dilated and the fœtal head was in the right occipito-anterior position. It was considered that the maximum diameter was still not quite through the brim. It was decided to allow labour to go on for three or four hours. An injection of one-sixth of a grain of morphine and one one-hundred-and-fiftieth of a grain of hyoscine was given. When the patient was seen at 4.30 a.m., four hours later, it was observed that the head was descending into the brim and a large caput was just visible *per vaginam*. At 8 a.m. a male child, weighing seven pounds one ounce, was born without assistance. There was an extremely large *caput succedaneum*, and pronounced moulding was present. The puerperium was uneventful.

Dr. Saltau's second patient was an Italian woman, a *primipara*, aged twenty-five years. She too was a small woman, her height being four feet eight inches. Her external measurements were: interspinous, nine inches; intercristal, ten and a quarter inches; external conjugate, seven inches. Her expected date of confinement was March 14, 1940. At term the head was unfixed. The membranes ruptured prematurely at 3.20 a.m. on March 19, 1940. Satisfactory contractions commenced shortly afterwards, and the patient was given one-sixth of a grain of heroin by hypodermic injection at 5.15 a.m. A vaginal examination at 10 a.m. revealed that the cervix was dilated sufficiently to admit three fingers, and the membranes had ruptured. The head was not movable; but the maximum diameter of the head was above the pelvic brim. At this stage the contractions subsided. The next morning, at 9.40, the maximum diameter was still above the brim, but the contractions remained infrequent and feeble. The next morning, forty-eight hours after the onset of labour, they became stronger and more regular. The head made no advance, and at 4 p.m. on March 21, 1940, vaginal examination revealed that the os was half dilated, the cervix was very thin and there was a large caput. Most of the head was still above the brim, and meconium was present. At 5 a.m. lower segment Cæsarean section was performed; the child was delivered alive. It weighed six pounds twelve ounces. Convalescence was uneventful.

Dr. Saltau said that he had shown these two patients to illustrate the use of a "trial of labour" at term when disproportion was evident. In the first case the outlook was dubious, and it was thought that Cæsarean section would very probably be necessary; but the contractions were so efficient that when a vaginal examination was made about twelve hours after the onset of labour the os was found to be three-quarters dilated. In the second case, in which the size of the pelvis was somewhat similar, the uterine function was deficient, as evidenced by the slow dilatation and failure of the head to advance after a 48-hour labour, although the baby was slightly smaller. Dr. Saltau stressed the difficulty in anticipating the outcome in these cases before the onset of labour. It was impossible to foresee the strength of the contractions in any one case, and other factors that had to be taken into consideration were the size of the baby, the time at which the membranes ruptured and the position of the head.

Dr. Saltau showed a third patient, a *primipara*, aged twenty-two years, who expected to be confined on July 2, 1940. She also was small in stature, being four feet ten and a half inches in height. The external measurements were: interspinous, eight and a half inches; intercristal, nine and a quarter inches; external conjugate, seven and three-quarter inches. She had had poliomyelitis two and a half years earlier, and there was still some residual paralysis of the abdominal muscles on the right side and of the flexors and adductors of the thigh. Dr. Saltau thought, however, that that would not complicate her labour; she was shown as another candidate for a "trial of labour" at term, when she would be watched closely.

(To be continued.)

Naval, Military and Air Force.

APPOINTMENTS.

THE undermentioned appointments, changes *et cetera* have been promulgated in the *Commonwealth of Australia Gazette*, Numbers 84 and 88, of May 9 and 16, 1940.

AUSTRALIAN IMPERIAL FORCE.

Australian Army Medical Corps.

To be Colonels.—Colonel N. L. Speirs, V.D., Unattached List, and to command 4th Australian General Hospital, 17th

April, 1940, and Lieutenant-Colonel (Temporary Colonel) A. P. Derham, M.C., Australian Army Medical Corps, 3rd Military District, and to be Assistant Director of Medical Services, Headquarters, Australian Overseas Base Sub-Area, 16th April, 1940.

To be Lieutenant-Colonel.—Lieutenant-Colonel J. K. Adey, O.B.E., Australian Army Medical Corps, 3rd Military District, and to command 2nd/1st Casualty Clearing Station, 4th April, 1940, *vice* Lieutenant-Colonel F. K. Norris, who relinquishes the command on 3rd April, 1940.

To be Major.—Major Joseph Hans Stubbe, Australian Army Medical Corps, 5th Military District, 13th October, 1939. (In lieu of notification respecting this officer which appeared in Executive Minute No. 12/1939, promulgated in *Commonwealth Gazette* No. 167, of 14th December, 1939.)

To be Captains.—Captains F. D. Stephens, Australian Army Medical Corps, 3rd Military District, 26th March, 1940, C. B. Cox, Australian Army Medical Corps, 2nd Military District, 20th March, 1940, and A. V. Jackson, Australian Army Medical Corps, 3rd Military District, 5th April, 1940.

2nd Reinforcements.—To be Captain—Captain W. H. Campbell, Australian Army Medical Corps, 2nd Military District, 27th March, 1940.

3rd and 4th Reinforcements.—To be Captains—Honorary Captains K. H. Tepper, Reserve of Officers (A.A.M.C.), 5th Military District, 5th April, 1940, and L. P. Sapsford, Reserve of Officers (A.A.M.C.), 1st Military District, 9th April, 1940.

To be Colonel.—Lieutenant-Colonel F. K. Norris, and to be Assistant Director of Medical Services, 7th Division, 4th April, 1940.

AUSTRALIAN MILITARY FORCES.

NORTHERN COMMAND.

First Military District.

Australian Army Medical Corps Reserve.

To be Honorary Captain.—Robert Andrew Douglas, 17th April, 1940.

EASTERN COMMAND.

Second Military District.

Australian Army Medical Corps Reserve.

To be Honorary Captains.—James Inglis Robertson and John Langston Watt, 12th April, 1940, and 16th April, 1940, respectively.

To be Honorary Captains.—Lieutenants J. M. McLachlan, C. S. Barham, J. S. Binns, A. A. Goodall, O. Bennett, G. D. Donkin, H. F. Thompson, F. C. Haddan, E. A. Lindsay, W. L. Medlicott, W. F. H. Smith, M. D. Hunter, S. J. Hogue, R. V. Considine and S. O. Fernon, 19th April, 1940; Honorary Lieutenants A. S. Cambage, M. S. Allan, K. C. T. Sutherland, T. G. Hall, E. H. Bestic, K. Y. Kerr, J. D. Hunt, E. S. McDermott, L. S. Beckett, N. Cope, L. J. Moxham, R. J. F. Gates, B. Briscombe, N. F. Crozier, G. H. Hogg, D. M. Whalen, E. P. Amesbury, J. T. Rogers, C. L. Burgess, F. R. Reid, F. E. Holt, S. L. Jackson, E. H. Harvie, A. G. Hunter, R. F. Arnold, H. A. Muller, T. M. Lloyd, G. E. Hawthorne, A. S. Henderson, C. F. Miller, E. J. McGee, H. D. Borthwick, A. C. Gabriel, J. H. Palmer, A. G. H. Lawes, T. M. Watson, S. J. Pogson, C. H. Hughes, C. R. Boge, K. C. D. Ward, R. W. F. Gates, R. F. Deck, R. Moore, A. A. Cooper, R. W. Halliday, L. M. D. Wall, M. D. Asplin, C. S. Denston, G. S. Cottee, C. C. Everingham, H. R. Campbell, C. A. French, R. B. Parker, R. P. Lane, K. B. Chisholm and R. W. Wright, 19th April, 1940. Honorary Captains D. D. Gibson and B. R. Jones are retired.

SOUTHERN COMMAND.

Third Military District.

Australian Army Medical Corps.

Captain A. D. Matheson is granted the temporary rank of Major whilst seconded as Deputy Assistant Director-General of Medical Services, 19th April, 1940.

Australian Army Medical Corps Reserve.

To be Honorary Captains.—Lieutenants J. N. Barker, B. J. Thompson, G. A. Payne, L. A. Freeman, W. S. Wilkinson, L. R. McGregor, E. J. Adam, G. B. McNair, W. J. Lumsden, F. T. Herbert, A. S. Campbell, A. E. B. Barnes, E. W. Wills, W. Adam, R. M. Gillies, B. S. Melrose, E. B. Adamson and V. S. H. Osment, and Honorary Lieutenants J. N. Rattray, D. Evans, L. V. Stephens, J. M. Lewis, R. D. McCallum, G. H. Fetherston, A. L. Beischer, A. R. Brown, P. Clark, B. T. Bell, A. J. J. Treacy, P. Pincus, G. M. Allen, W. M. Eggleston, R. W. Towns, W. T. C. Thomas, R. B. C. Campbell, G. A. J. Parrett, J. H. Lane, F. I. Hicks, H. E. Krutli, P. R. N. Sutton, R. G. Morris, B. M. Challen, J. K. Herd, P. B. Malcom, E. C. Bailey, F. Nilson, A. G. Parker, F. Williams, C. H. K. Douglas and W. H. Beischer, 4th April, 1940.

Fourth Military District.

Australian Army Medical Corps Reserve.

Honorary Captains L. J. Pellew and R. E. Magarey are retired.

To be Honorary Captain.—Honorary Lieutenant A. J. Bloomfield, 21st December, 1939.

WESTERN COMMAND.

Fifth Military District.

Australian Army Medical Corps.

To be Major (temporarily).—Captain (provisionally) G. R. Troup, with regimental seniority next after Major (temporarily) R. G. Williams, 4th April, 1940.

Honorary Captain K. D. Gray is appointed from the Reserve of Officers (A.A.M.C.), and to be Captain (provisionally), 14th December, 1940. (In lieu of notification respecting this officer which appeared in Executive Minute No. 18/1940, promulgated in *Commonwealth Gazette*, No. 65 of 11th April, 1940.)

Australian Army Medical Corps Reserve.

To be Honorary Captain.—Daniel Alphonsus Quinlan, 11th April, 1940.

ROYAL AUSTRALIAN AIR FORCE.

Citizen Air Force.

Medical Branch.

Henry David Phipps, M.B., B.S., is granted a commission on probation with the rank of Flight Lieutenant with effect from 9th April, 1940.

John Felix Frayne, M.B., B.S., is granted a commission on probation with the rank of Flight Lieutenant with effect from 13th April, 1940.

The following are granted commissions on probation with the rank of Flight Lieutenant with effect from 1st May, 1940: Reginald Terrence McCaul, B.D.S., Rex Grose Plummer, M.B., B.S., and Reginald James Stabback, M.B., B.S.

Flight Lieutenant K. E. Rex, M.B., B.S., is transferred from the Reserve to the Active List, 17th April, 1940. The notification in the *Commonwealth of Australia Gazette*, dated 14th March, 1940, concerning Lionel Pelham Sapsford, M.B., B.S., is cancelled.

Reserve.

Medical Branch.

The following are granted commissions on probation with the rank of Flight Lieutenant with effect from 1st May, 1940: Mervyn Henry Bowzer Robinson, M.B., B.S., F.R.C.S., F.R.A.C.S., Denis Thorman Shortridge, M.B., B.S., and Harold Chisholm Worch, M.B., B.S.

Flight Lieutenant W. W. Ingram, M.D., F.R.A.C.S., is granted the honorary rank of Squadron Leader with effect from 1st May, 1940.

The notification in the *Commonwealth of Australia Gazette*, dated 7th March, 1940, concerning Herbert Malcolm Franklands, M.B., B.S., D.T.M., is cancelled.

Flight Lieutenant M. L. Powell, M.B., B.S., M.R.C.P., relinquishes his commission with effect from 30th April, 1940. (Ex. Min. No. 25—Approved 8th May, 1940.)

Medical Societies.

THE MEDICAL SCIENCES CLUB OF SOUTH AUSTRALIA.

A MEETING of the Medical Sciences Club of South Australia was held at the University of Adelaide on November 3, 1939.

The Sulphur Metabolism of Plants.

PROFESSOR J. G. WOOD read a paper entitled "Sulphur Metabolism of Plants". He dealt with work on sulphur metabolism of plants carried out by Miss B. S. Barrien and himself. The main facts presented were as follows.

1. During the life cycle of an annual grass (*Andropogon sudanensis*) the absolute and relative amounts of protein sulphur in the leaves rose to a maximum and thereafter decreased. The higher the level of nitrogen manuring, the higher was the content of protein sulphur at any time. During the life cycle the ratio of protein nitrogen to protein sulphur progressively decreased and suggested changes in amounts of individual proteins with age.

2. During starvation protein sulphur decreased in amount in leaves and was accompanied by an equivalent increase in inorganic sulphate sulphur. No ethereal sulphate could be detected. The inorganic sulphate accumulated in the leaves and did not migrate to the stems and roots as did amino-acids and amides. No change in protein sulphur or sulphate sulphur occurred in stems and roots.

3. Increased manurial doses of potassium sulphate caused an increase in sulphate in the leaves, but no change in the amounts of protein sulphur or free cystine.

4. Varying doses of cystine applied to the soil in which plants were growing resulted only in an increase of inorganic sulphate, but no change in protein sulphur or free cystine occurred.

5. Increased doses of ammonium salts caused an increase in protein sulphur and free cystine.

Possible mechanisms for the course of the reactions concerned were discussed.

Glycosuria in South Australia.

DR. RAY HONE read a paper entitled "Some Observations on Glycosuria in South Australia". He introduced his subject with a brief account of the history of sugar in the urine. He pointed out that during this century it had been recognized increasingly that in a certain number of cases of glycosuria the condition was not diabetic, but of an innocent nature. In fact, the diagnosis in a case of glycosuria had come to rest mainly on the findings of a sugar tolerance curve.

Dr. Hone then showed specimens of the different types of sugar tolerance curves, and showed how the type of response could be varied by preceding diet, infection *et cetera*. He showed further examples of errors that could occur in the plotting of curves or in their interpretation through faulty technique.

Finally, he gave the figures for a series of cases investigated here over a period of ten years. This showed that in this State one case in three of glycosuria was of an innocent nature. This was a very high percentage of innocent cases compared with figures quoted from America and England. He invited discussion on this point as to whether anything in the different environment in South Australia could account for this high proportion.

Cinematograph Films.

A colour film taken at Channell Island Leper Station, and another more general one, depicting scenes in northern Australia, were shown by Mr. H. A. Behrens.

Correspondence.

PATHOLOGY AND BLOOD TRANSFUSION.

SIR: The article on "Recent Advances in the Pathology of Blood Transfusion" by Dr. Noel Gutteridge, which appeared in THE MEDICAL JOURNAL OF AUSTRALIA of April 27, 1940, will, we think, be read with approval by all those interested in the subject, who will appreciate the clarity with which various new developments have been described by the author.

There is one aspect, however, on which we think comment is desirable. Dr. Gutteridge omits any specific reference to the transfusion services already existent in Australia, and states: "With the increased employment of blood transfusions as a therapeutic measure it is found that the demand for blood has exceeded the supply of charitably minded donors . . ."

We would like to point out that a Voluntary Blood Transfusion Service, organized by the Victorian Division of the Australian Red Cross Society, on the lines of the London service, has successfully operated in Melbourne since 1929. More recently a similar service has been formed by the Red Cross Society in Perth, Western Australia, and in some of the other States voluntary transfusion services have been established by other associations, such as "Toc H." In Melbourne during these ten years, in spite of steadily increasing demands, the supply of "charitably minded" persons willing to act as voluntary donors has never failed us. Occasionally our numbers have fallen a little short; but at such times fresh appeals have quickly resulted in a generous response. During the latter part of this period, in fact, it has rarely been necessary to make such appeals, as a steady stream of volunteers have come forward from among the friends of preexisting donors, who, when treated with consideration and shown that their public-spirited service is appreciated, are often most enthusiastic and effective "propagandists".

Since the outbreak of the war the Victorian Division of the Red Cross Society has also organized an Emergency Blood Transfusion Service. The volunteers for this service (most of whom are women), if found suitable as regards physique and veins, are asked to act, firstly, as donors to replace those on the regular list who have become unavailable by reason of enlistment in the Australian Imperial Force or militia service, and secondly, for special war-time needs, such as the provision of large quantities of high-titre agglutinating serum (prepared at the Commonwealth Serum Laboratories) necessary for the efficient performance of the blood group tests which are being carried out on the entire personnel of the Australian Imperial Force, and of stored serum for therapeutic use.

The publicity given to the need for donors for this emergency service has also resulted in a gratifying increase in volunteers for the permanent service, among people who have stated that they were previously unaware, or only vaguely aware, of its existence and purpose.

We whole-heartedly agree with Dr. Gutteridge in attributing the success of the London service very largely to the personality of its honorary secretary, Mr. P. L. Oliver, who, with Dr. Brewer, has so ably pioneered and maintained this vast undertaking. We feel justified, nevertheless, in view of our favourable experience with the volunteers in our own city and the cooperative spirit shown by the hospitals and medical practitioners using the service, in contending that the London service need not necessarily be regarded, as Dr. Gutteridge would seem to infer, as an exception proving a rule that professional services are to be preferred to, or are more likely to be successful, at any rate in this country, than voluntary ones, provided, of course, that the latter can be conducted on suitable principles, such as those governing the London service.

Dr. Gutteridge writes that "even Mr. Oliver states, however, that he has now gained a clearer idea of the arguments in favour of the paid donor principle". In

the British Red Cross Society Blood Transfusion Service quarterly circular for January, 1938, in which he describes his visit to the second International Blood Transfusion Conference held in Paris in September, 1937, at which he met many Continental protagonists of the professional system, Mr. Oliver does use such words; but their context is as follows: "Whilst more firmly convinced than ever as to the immeasurable superiority of voluntary as against professional blood donors, I feel bound to admit that I have now gained a clearer idea of the arguments in favour of the latter principle." These arguments, as set out in this circular by Mr. Oliver (in too great detail for quotation here) are not, in our opinion, very inspiring or convincing. Victor Riddell, F.R.C.S., in his book "Blood Transfusion" (Oxford University Press, 1939) gives another interesting and instructive analysis of the advantages and disadvantages of the two systems, voluntary and professional, which, although admittedly also based on experience of the London and other British voluntary services, appears to us to provide further argument in favour of the voluntary system.

Those who are familiar with our service will know that although for many years it was reserved for the use of the public hospitals, more latterly it has accepted calls for private patients, who may be just as greatly in need of prompt transfusion as those in public hospitals, and who are sometimes without friends or relatives suitable to act as donors. At the time when the service was first made available for private patients, it was considered advisable to establish for these patients a professional section of the service, for which a nucleus was formed by university students (who have traditionally acted as professional donors for private patients in this city), but which other prospective donors also had the right to join in preference to the voluntary section, if they desired. The Voluntary Donors' Association, however, recently strongly urged that its members be allowed to act for private patients; they guaranteed to maintain a strength of personnel sufficient to enable them to do this without interfering with the claims of the public hospitals, and suggested that, in lieu of fees to themselves, payments by private patients should be made to the funds of the service. The professional section is therefore being discontinued, and private patients for whom voluntary donors are supplied, are asked to make donations, according to their means, in place of the fees required for professional donors, towards the maintenance expenses of the service.

We would like to pay a final tribute to the spirit of our voluntary donors by referring to an incident which occurred at a meeting of donors held in 1938. At this meeting the question of professionalism was discussed in relation to the proposed formation of a voluntary donors' association. During the discussion a voluntary donor of many years' standing rose and spiritedly denounced the acceptance of "blood money"; he was received with tumultuous applause, the meeting voted in favour of the continuance of the voluntary system, and an enthusiastic Voluntary Donors' Association came into being.

Yours, etc.,

J. NEWMAN MORRIS,
Chairman,
Victorian Division, Australian Red Cross Society.

LUCY M. BRYCE,
Honorary Medical Officer,
Red Cross Blood Transfusion Service.

Melbourne,
May 15, 1940.

WHOOPING COUGH IN MELBOURNE.

SIR: There is in Melbourne at present a moderately severe outbreak of whooping cough, and it would seem useful at this stage to raise the question of prophylactic vaccination in this disease.

Work at the Children's Hospital, Melbourne, carried out under the auspices of the National Health and Medical Research Council, definitely supports the claims of overseas observers, that a suitable vaccine administered in adequate dosage will protect pertussis contacts from developing the disease, providing the early catarrhal cough has not made its appearance. Indeed in some cases, even if this cough has already been present for a few days, the administration of the vaccine would appear to lessen the severity of the attack. If, however, the disease has entered upon its fully developed phase, treatment with vaccine is useless.

One still meets cases where parents of contact children have been medically advised against the prophylactic administration of vaccine. It is the purpose of this letter to point out that such advice may allow the unnecessary development of the disease—a disease which, it is hardly necessary to state, carries a high mortality in infants and condemns older children to several weeks of incapacitating and extremely uncomfortable cough.

Cases of chronic bronchitis and bronchiectasis with an antecedent history of whooping cough are seen so frequently in the out-patient department of the Children's Hospital that the seriousness of this disease cannot be overstated. So may I be permitted, sir, to urge through your columns, the attention of all practitioners to the available simple procedure in the prophylaxis of this disease.

The vaccine used in the above-mentioned work is that prepared from local strains of the Bordet-Gengou bacillus by the Commonwealth Serum Laboratories, and supplied as "Phase I Pertussis Vaccine, Strength C" (10,000 million organisms per cubic centimetre), total dosage given being a minimum of 8·0 cubic centimetres, in a series of five injections, commencing with 0·5 to 0·75 cubic centimetre, depending on the age of the child and the urgency of the case. No severe or serious reactions, either local or general, have been observed.

Yours, etc.,

DOROTHY GEPP.

Melbourne,
May 16, 1940.

HYDATID DISEASE OF THE PELVIS AND FEMUR.

SIR: I have received from Professor J. B. Cleland a letter in reference to my article "A Proctological Problem" in this journal of April 6, in which he states: "Unfortunately, with this title, your case is not likely to be indexed under Hydatid Disease, and consequently will be lost to those in search of such cases."

I am grateful to Professor Cleland for his kindness in drawing my attention to the likely effect of my own title, which would be remedied by your publication of this letter.

Yours, etc.,

G. M. OXER.

Royal Australian Air Force,
Point Cook,
Victoria.
May 3, 1940.

MODERN METHODS IN PÆDIATRICS.

SIR: I have read with much interest the address on modern methods in pædiatrics delivered by Dr. F. Arden, of the Brisbane Children's Hospital. It recalls to me the three years which I spent long ago as resident in the same hospital, followed by thirty years as an honorary.

Firstly, let me express my pleasure at his recognition of the importance of nutrition in the ailments of children, and in particular of his recognition of the fact that many children suffer from a deficiency of vitamin *B* in their diet, second only in importance to a deficiency of milk. There are two methods of combating nutritional deficiency: firstly, by adding supplements to the diet; secondly, by a radical improvement in the diet itself. I hope Dr. Arden

does not restrict himself to the first method. Wheat germ supplements are no doubt valuable, but their use is only temporary. After the child has recovered his health and returned home, he will almost certainly be put back on his old diet. Judging from personal experience, I have long feared that hospitals will be the last places where anyone can obtain a rational diet. Now I am beginning to hope that good feeding in hospital may be an example that will have some effect on parental ideas about food. This example would be intensified if hospital nurses and even the medical staff would cease eating devitaminized bread, which during the past eighty years has had such a detrimental effect on national health.

I am interested in the problem of threadworms. They are common and occur in apparently healthy as well as in sickly children. The *Oxyuris* cannot propagate in the bowel, nor does it lay its eggs there. It crawls outside the anus to do this, causing the nocturnal itching that is so familiar. Anthelmintics will dislodge most of them, but commonly a few remain. By rubbing his itching seat the child covers his fingers with their ova, many being lodged under the finger-nails. He then proceeds to reinfect himself by putting his fingers into his mouth, and may infect also his brothers and sisters, even the baby, in the same way. When young women suffer from threadworms I have always found that they have the habit of biting their nails. I note that the old idea that threadworms live only in an unhealthy bowel is being revived. It is possible; but it needs very strict proof. Several possible fallacies may be suggested. Whenever a child is ailing without evident cause, someone suggests that he is suffering from worms. Therefore in these children special search is made. No doubt many children receive "worm powders" from chemists and, I fear, from some medical practitioners, for worms that they have not got; but in others the worms are looked for and found. No threadworm survey is made of healthy children A second possibility is that unhealthy children are more sensitive to anal itching, and a third that they are more likely to bite their nails. Finally, it is never proved that a child is cured of threadworms unless his motions are carefully examined three months after treatment.

Great victories have been won in the treatment of acute disease in children since I was resident. I well remember my desperate attempts to resuscitate those *in extremis* from infection, and their almost invariable failure. In a few instances subcutaneous or intravenous salines did appear to have a marvellous effect, but as a rule this was only temporary. The modern methods of intravenous injections, and especially those of blood transfusion are immensely superior. All hope of combating coccal infections by therapeusis had, after innumerable attempts, been in my time completely abandoned. The introduction of sulphapyridine and its congeners has changed all this. These weapons have made a tremendous increase in our responsibilities for treatment. To fail to use them rightly is fatal.

Dr. Arden objects to the preliminary dose of castor oil in the treatment of diarrhœa on account of the bowel having been already emptied before it is given, and because it may be an irritant. When I was a resident I made *post mortems* on infants dead from acute toxæmic diarrhœa, and was much struck by the fact that, though the large bowel was empty, the small bowel contained much fæcal matter. So the first objection is unsound. As for the second, I believe that castor oil is frequently placed on the conjunctiva without causing irritation, and I cannot therefore believe that it is more irritating to the bowel than food undergoing bacterial decomposition. The first dose should not be repeated. In the early convalescent stage I have found whey containing no more than a minute trace of casein and fat, but all the other soluble constituents of cow's milk, inferior only to breast milk, and far superior to malted, skimmed or condensed milk.

I must pass over other interesting points raised by Dr. Arden, but I cannot omit my congratulations on his use of the apparent discovery of a simple diagnostic method for estimating the activity of rheumatic infections. If trustworthy, it is most important; but here also I think very strict proof is necessary.

In conclusion I must make a few comments on Dr. Arden's diet sheet for babies. My first reaction was to murmur: "This is a diet *de luxe*; can we apply it to the twenty thousand babies born annually in Queensland?" This was followed by the comforting thought that most of them are, or ought to be, on the breast. True, mother's milk is often deficient in vitamin *B*; but by putting the mother on "Cerevite" or wholemeal bread, to the total exclusion of white bread, we easily cure mother and child. Mother's milk may be deficient in iron—probably not so frequently here as in Britain; but this should not occur on a healthy diet, unless she has suffered an excessive blood loss. For an artificially fed infant there are easy ways of administering iron for a few months without unduly complicating its diet. Frankly, I have a horror of hard and fast diet schemes for infants, and Dr. Arden's neat little chart aroused this in full blast. In our teaching we have to outline some average diet, but the artificial feeding of infants is a field in which we must be guided by experience and general principles only. In each individual case we should carefully feel our way like a blind man in a railway carriage. For instance, I have known cases in which the early introduction of starchy food has been useful; but they have been very few. Those in which it has been unnecessary or harmful have been many. The same objection applies to the early introduction of egg yolk, broth *et cetera*, and the risk of sensitization to egg albumen is not imaginary. I know that at present there is a reaction towards these old methods, and we have had opportunities of observing its harmful effects.

Dr. Arden has apologized quite unnecessarily for the length of his most interesting address. I will ask your readers to endure the tediousness of an old man who still imagines himself youthful and enthusiastic.

Yours, etc.,

A. JEFFERIS TURNER, M.D., D.P.H.,
Late Director of Infant Welfare in Queensland.

Brisbane,
May 18, 1940.

Post-Graduate Work.

LIBRARY SEMINARS AT SYDNEY.

LIBRARY SEMINARS and clinico-pathological conferences, arranged by the Directors of Medicine, Surgery and Pathology, are held each month at the Prince Henry Hospital, Little Bay. These meetings take place at 4.30 p.m. on the second and fourth Mondays in the month, public holidays excluded. In the meetings, which are quite informal, special attention is given to recent literature, and it is the aim of the Directors to encourage free discussion on the selected subjects. A cordial invitation to be present is extended to all medical practitioners.

The next library seminar will be held on Monday, June 10, 1940. Subject: "Cerebral Hæmorrhage".

Proceedings of the Australian Medical Boards.

QUEENSLAND.

THE undermentioned have been registered, pursuant to the provisions of *The Medical Act of* 1939 of Queensland, as duly qualified medical practitioners:

Cummine, Harold George, M.B., B.S., 1939 (Univ. Sydney), Thursday Island.

Riddell, Rees James, M.B., B.S., 1935 (Univ. Melbourne), D.T.M. and H., R.C.P. and S. (London and England), 1939.

Grigor, William Ernest, M.B., Ch.M., 1908 (Univ. Sydney), Tara.

The Royal Australasian College of Physicians.

EXAMINATION FOR MEMBERSHIP.

THE examination for membership of the Royal Australasian College of Physicians will be held in Sydney in August and September, 1940.

By-law 10, Section (2), states:

The examination for Membership of the College shall consist of:

(1) A paper on the Principles and Practice of Medicine, including Pathology, Therapeutics and the History of Medicine.

(2) An oral examination, which may include the clinical examination of patients, together with the identification of naked-eye and microscopic specimens.

The written paper will be taken in capital cities where candidates are offering, on Saturday, August 3, 1940, and the clinical examination will be conducted in Sydney on Wednesday and Thursday, September 4 and 5, 1940.

Application forms may be obtained from the office of the College, 145, Macquarie Street, Sydney, and should be in the hands of the acting honorary secretary at this address not later than July 6, 1940.

Nominations and Elections.

THE undermentioned has applied for election as a member of the New South Wales Branch of the British Medical Association:

Johnson, Adrian Mackey, M.B., B.S., 1939 (Univ. Sydney), 2/5 Field Regiment, A.I.F., Ingleburn.

Diary for the Month.

JUNE 4.—New South Wales Branch, B.M.A.: Organization and Science Committee.
JUNE 5.—Western Australian Branch, B.M.A.: Council.
JUNE 5.—Victorian Branch, B.M.A.: Branch.
JUNE 6.—South Australian Branch, B.M.A.: Council.
JUNE 7.—Queensland Branch, B.M.A.: Branch—Joseph Bancroft Memorial Lecture.
JUNE 11.—Tasmanian Branch, B.M.A.: Branch.
JUNE 11.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
JUNE 14.—Queensland Branch, B.M.A.: Council.
JUNE 18.—New South Wales Branch, B.M.A.: Ethics Committee.
JUNE 19.—Western Australian Branch, B.M.A.: Branch.
JUNE 20.—New South Wales Branch, B.M.A.: Clinical meeting.
JUNE 25.—New South Wales Branch, B.M.A.: Medical Politics Committee.

Medical Appointments Vacant, etc.

For announcements of medical appointments vacant, assistants, *locum tenentes* sought, etc., see "Advertiser", pages xviii-xxi.

ADELAIDE CHILDREN'S HOSPITAL, ADELAIDE, SOUTH AUSTRALIA: Honorary Officers.
COMMONWEALTH DEPARTMENT OF HEALTH: Medical Officers.
COMMONWEALTH OF AUSTRALIA: Medical Officer.
PERTH HOSPITAL, PERTH, WESTERN AUSTRALIA: Resident Pathologist.
RENWICK HOSPITAL FOR INFANTS, SUMMER HILL, NEW SOUTH WALES: Resident Medical Officer.
ROYAL ALEXANDRA HOSPITAL FOR CHILDREN, SYDNEY, NEW SOUTH WALES: Honorary Officers.
ROYAL PRINCE ALFRED HOSPITAL, SYDNEY, NEW SOUTH WALES: Honorary Officers, Acting Bacteriologist.
ST. GEORGE DISTRICT HOSPITAL, KOGARAH, NEW SOUTH WALES: Honorary Assistant Orthopædic Surgeons.
SYDNEY HOSPITAL, SYDNEY, NEW SOUTH WALES: Temporary Honorary Relieving Assistant Physician.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment referred to in the following table without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

BRANCHES.	APPOINTMENTS.
NEW SOUTH WALES: Honorary Secretary, 135, Macquarie Street, Sydney.	Australian Natives' Association. Ashfield and District United Friendly Societies' Dispensary. Balmain United Friendly Societies' Dispensary. Leichhardt and Petersham United Friendly Societies' Dispensary. Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney. North Sydney Friendly Societies' Dispensary Limited. People's Prudential Assurance Company Limited. Phœnix Mutual Provident Society.
VICTORIAN: Honorary Secretary, Medical Society Hall, East Melbourne.	Associated Medical Services Limited. All Institutes or Medical Dispensaries. Australian Prudential Association, Proprietary, Limited. Federated Mutual Medical Benefit Society. Mutual National Provident Club. National Provident Association. Hospital or other appointments outside Victoria.
QUEENSLAND: Honorary Secretary, B.M.A. House, 225, Wickham Terrace, Brisbane, B.17.	Brisbane Associate Friendly Societies' Medical Institute. Proserpine District Hospital. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL or position outside Australia are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.
SOUTH AUSTRALIAN: Secretary, 178, North Terrace, Adelaide.	All Lodge appointments in South Australia. All Contract Practice Appointments in South Australia.
WESTERN AUSTRALIAN: Honorary Secretary, 205, Saint George's Terrace, Perth.	Wiluna Hospital. All Contract Practice Appointments in Western Australia.

Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

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